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VOLUME V

1911-1912

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MEDICAL AND SURGICAL

REPORT

OF

Bellevue and Allied Hospitals

IN

THE CITY OF NEW YORK.

VOLUME V.

1911-1912.

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A CASE OF OSTEITIS DEFORMANS.*

GUY WALLACE, M.D.

The case of Paget's disease is one from Dr. Van Horne Norrie's service at Bellevue Hospital. The patient was admitted November 4th, 1911, dying seventeen days later, and presented the essential manifestations of this disease.

The clinical history is very meagre. The man had been a heavy drinker and smoker; gave a history of chancre, without secondaries, had gonorrhea four times; was a veteran of the Civil War, and during the past twenty years had occupied a position as salesman; during the last two years he had been living on his pension. The patient came to the hospital because he was becoming weaker and was unable to take care of himself. A diagnosis of Paget's Disease was made from the massive head. There was but a slight bowing of the right femur, the hands and feet being normal. There was an indistinct history of muscular rheumatism during the previous year. The patient stated that when fifty years of age he wore a No. 7 hat, and that practically every four years since that time he required a larger one. His picture in uniform, taken at the time of the war, shows no enlargement of the face or head. This cranial enlargement, apart from the sensation of weight, caused him no discomfort.

The chief complaints on admission were, a dull aching pain in the back, weakness and headache. During his stay in the hospital he developed flaccid paralysis of the left arm, with flattening of the left side of the face, and deviation of the tongue to the left. The radials and temporal arteries were extremely prominent, being practically converted into tortuous pipe-stems. The Wasserman reaction was negative. Broncho-pneumonia supervened and, after having been in coma with stertorous respiration for two days, he died.

The autopsy was performed by Dr. Charles Norris the following day and the anatomical diagnosis was as follows:

Cerebral hemorrhage.
Paget's disease.
Chronic and acute pachymeningitis hemorrhagica fibrinosa.
Marked pial oedema.
Extreme arterio-sclerosis of cerebral and coronary arteries.
Chronic emphysema and bronchitis.
Lobular pneumonia.
Acute localized fibrino-purulent pleuritis, left.
Acute fibrinous pleurisy, right.
Slight chronic intestitial and acute parenchymatous nephritis.
Enlarged prostate.
Hypertrophy of the lingual follicles.

Body is that of an old man 70 years of age, 5 feet 6 inches in height. There is considerable emaciation of the extremities and trunk. Thorax is moderately long, conformation normal. There is slight bowing of the legs. The knee joints are of normal conformation. The feet, toes, hands and fingers are normal. Breadth of hand 8 cm. The wrist, elbow, shoulder joints and vertebral column are normal. There is abundant dark hair over the chest; the pubae are abundant,

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masculine in type; there is considerable perineal hair; small amount of hair over thighs, axillary hair abundant. The hair partly gray, is abundant on the sides and scanty over the top of the head; eyebrows are dark and moderately thick; long grayish beard and moustache. The costal cartilages are of normal breadth. The first costal cartilage is calcified, the other cartilage being slightly greenish yellow on section with only a few calcific foci. The ribs are fragile. Intercostal spaces, ribs and cartilages are of normal breadth. Marrow of the ribs is abundant and dark red in color. The liver extends about a hand's breadth below the costal arch. Diaphragm is at the fourth space right and left.

Head: Head is very large and heavy, the face being normal in size. The temporal ridges are large and prominent; the temporal muscle is broad but thin. Tissues of the scalp are thin, as if stretched. The scalp peels readily. The pericranium strips readily. The external surface of the bone is everywhere slightly roughened, hyperaemic and irregular. The parietal foramina are very large and deep; similar accessory foramina are present. The surface is irregularly mottled with pale yellowish areas and reddish areas of hyperaemia. The sutures are everywhere obliterated. Conformation of the calvarium is somewhat irregular, but the symmetry of the skull, as a whole, is maintained. The increase in size of the skull is symmetrical, the calvarium being greatly thickened. The dimensions of the skull following removal of the scalp, were as follows:

Greatest circumference	65.5 cm.
Greatest diam. ant. post	23 "
Bi-parietal	18 "
Bi-malar	13.3 "
Naso-mental	14.2 "
Mento-coronal (length from the chin to the top of skull)	26.5 "
Occipito-frontal	22 "
From zygoma to top of calvarium	12.7 "
Length of the nose	6.5 "
Distance from angle of jaw to symphysis menti	6 "
Anterior fossa, greatest breadth (internally)	10.5 "
Anterior fossa, ant.-post	6.3 "
Middle fossa, breadth	14.8 "
Middle fossa, ant.-post	5 "
Breadth of occipital fossa	12.5 "

The forehead is massive. The wings of the sphenoid are not thickened. The infundibulum of the pituitary is long and reddish in color. The Foramen Magnum is funnel-shaped, 2 cm. in breadth. The Clivus Blumenbachii is approximately 5.6 cm. in length 4.4 cm. in breadth. The pituitary gland measures 18 cm. by 12 mm. by 3 mm. Its right lobe is hemorrhagic. The gland is pale yellowish in color and rather firm.

The middle ears are normal. The sphenoidal sinuses are large. The mucous membrane is normal. The dura is adherent, especially on the right side, where it tears on stripping. It is everywhere thickened and the inner surface shows an extensive hemorrhagic membrane formation over the cerebral convexities. The pia is markedly oedematous and the Pachionian granulations are not prominent. A number of small opaque foci, the largest 2 to 3 mm. in diameter, are found in the pia over the vortex, near the longitudinal fissure. There is a considerable amount of blood stained fluid at the base. The brain, after the ventricles were opened, weighs 1440 gm. On the superior surface of the cerebellum there is fresh, reddish blood clot, and there is a considerable hemorrhagic infiltration in the pial meshes along the posterior border of the right lobe of the cerebellum. The vertebrals are markedly sclerotic. The basilar artery is very large, extremely sclerotic, with intense yellowish patches. The arteries of the Circle of Willis and the cerebral arteries are markedly sclerotic. The smaller vessels show extensive nodular sclerosis. The left lateral ventricle is filled with blood stained fluid. The right lateral ventricle contains reddish blood

clot. There is an extensive hemorrhagic softening and infiltration involving the posterior and superior half of the lenticular nucleus, which extends into the optic thalamus and the adjacent wall of the third ventricle. The velum interpositum is infiltrated with blood and does not appear to be thickened. Otherwise the cross sections of the brain reveal no gross lesions.

The pineal gland measures 7 to 8 mm. laterally by 5.6 ant. post. The sella turcica is 28 mm. in breadth by 18 mm. ant. post. The clinoid precesses are enlarged. The calvarium is soft, sawing through readily. The thickness of the occipital bone is 2 cm., that of the frontal bone 1.5 cm. The diploe are noticeable only in the occipital bone.

Lungs: The pleural cavities are free from fluid or adhesions with the exception that the left apex is slightly adherent. The pleurae of both lungs feel sticky. The right lung is slightly emphysematous, the surface being roughened and irregularly mottled. An extra fissure, 2 inches in length by 1 inch in depth, is present in the lower lobe. The larger and smaller bronchi contain mucopurulent exudate and the mucous membrane is markedly congested. Section shows an extensive lobular pneumonia involving all lobes and more marked posteriorly. The left lung is rather voluminous; it does not feel heavy; is well aerated; the surface is smooth and on section shows nothing abnormal, with the exception of slight congestion. The pulmonary vessels are normal except for a few areas of intimal thickening. Bronchial lymph nodes are small and anthracotic. There is no evidence of a tuberculous process either in the lungs or in the lymph nodes.

Heart: The pericardium is normal and contains a small amount of clear fluid. The heart is normal in size, the apex being formed by the left ventricle. The vessels are slightly tortuous and show a few areas of nodular sclerosis. The right auricle is normal. Fossa ovalis is normal in size. The foramen ovale is patent. Endocardium is slightly opaque. Tricuspid valve measures 13 cm., and is normal. There is no hypertrophy or dilatation on the right ventricle. The mitral ring shows a slight narrowing, the aortic segment being contracted and calcified at its base. The wall of the left ventricle measures 25 mm. in thickness; the musculature is pale brownish red in color and on section shows numerous small areas of fibrosis. The aortic cusps are slightly thickened and calcified at the line of their attachment; the line of closure is, however, normal. The portion of the aorta below the first part of the arch is the seat of a slight patchy sclerosis. The arch, thoracic and upper part of the abdominal aorta being normal. The branches of the abdominal aorta are only slightly sclerotic. The lower half of the abdominal aorta and the iliacs are somewhat broad but show only a slight sclerosis.

Spleen: Is small, rather firm, weighs 100 gm. and measures 10½ x 4½ x 4 cm. On section it is congested and shows a slight increase in interstitial tissue.

Pancreas: Is about normal in size; lobulations are normal.

Liver: Weighs about 1600 gm. The serosa is normal and pale brownish in color. The surface is smooth and on section the lobulations are indistinct, otherwise normal

Gall Bladder: Is long and contains numerous faceted stones, there is a very large number present. Common bile duct normal. Patency was not tested.

Adrenals: Left adrenal measures 5½ cm. in length by 2½ cm. in breadth. The right adrenal measures 6 x 4 cm. The organs are somewhat firm. The cortex is pale yellowish in color and measures 2 to 3 mm. in breadth. The medulla is dark red in color and shows a few patches of glistening tissue.

Kidneys: The right kidney 120 gm., left 160 gm. They are somewhat firm, apparently normal in size. The capsules are adherent in a few places. The surface is slightly granular. Signs of fetal lobulations persist. There are a few superficial scars on surface. The cortex is thickened and pale, the markings are distinct. The pelves, ureters and urinary bladder are normal. The prostate is large; otherwise normal. The testicles and epididymes are normal in size and appearance; there is a small hydrocele on left side.

Oesophagus: Shows a few distended veins in its upper half; otherwise normal.

Stomach: The mucous membrane is congested. There is a small, whitish area situated beneath the cardia of the mucous membrane (Lymphangioma).

Intestines: Small and large intestines contain a small amount of gas and hard fecal material. Appendix is normal. In the coecum there is a small, reddish focus, 3 x 4 mm., which is freely moveable. There are several pale yellowish areas in the wall of the jejunum (Lymphangiomata). The mesentery is somewhat fatty. The lymph nodes are abundant but small.

Organs of Throat and Neck: The tongue is apparently normal in size. The lingual follicles are increased in size and number. There are a number of pale, elevated areas at the back and sides of the tongue, which on section show a milky fluid. The pharynx and larnyx are normal. The tonsils are enlarged, the right showing a number of depressions filled with exudate. The thyroid gland is somewhat symmetrically enlarged and pale reddish on section. The organ was not weighed. The parathyroid glands were removed.

The following measurements were made of the pelvis:

Intercristal	27 cm.
Interspinous	23.3 "
Internal conjugate	11.2 "
From the greater trochanter to plane of surface of foot	34.5"
From ant. sup. spine to plane of surface of foot	37.4"
Ant. sup. spine to bottom of external condyle of femur	19"

The right knee joint is normal as to its articular surface and mucous membrane. The right femur is bowed. It is broad in its lower half and measures 18 cm. immediately above the condyles. On section the cortex measures 1 cm. in diameter. The diameter of the bone is 3.1 cm. The cortex of the left femur measures 6 mm. The diameter of the whole bone is 3.1 cm., right and left being of the same diameter. At a place above the middle of the right tibia, the thickness of the cortex is 1 cm. The bone is very firm, almost ebony-like. Vertebrae and marrow everywhere normal.

Ribs: The marrow is red in color and normal in amount.

One of the earliest terms used in referring to diseases of bones is rickets. This term was used to include all diseases in which extensive bone changes occurred. Since then, groups of cases have been separated, such as Infantile Rickets, Achondroplasia, etc. The term Osteitis Deformans has long been used in connection with various diseases, which resulted in deformity of the affected bones. This term included constitutional diseases, fractures, and congenital deformities. The term was first used by Czerny in 1873 to describe a case of spontaneous curvature of the lower limbs. (This was in all probability a case of Osteomalacia.)

Sir James Paget who, in 1876, made use of the term Osteitis Deformans, in describing the condition which now bears his name, remarks in his original article: "Holding then the disease to be an inflammation of the bone, I would

suggest that, for brief reference and for the present, it may be called after its most striking character, osteitis deformans. A better name may be given when more is known of it."

Since that time various names have been applied to conditions apparently analogous to that described by Paget. To Paget's (1) original description, which appeared in the Medico-Chirurgical Transactions, 1876, little has been added. As he described it, the disease begins in middle life or later, progressing slowly during a period of many years, and causing no disturbances other than those due to mechanical changes in the diseased bones, those most often involved being the long bones of the lower extremity, the cranium, spine and clavicles. These enlarge and soften, and, owing to the pressure of the body and muscular traction, become curved and misshapen so that the statue steadily decreases. In a later communication, he states that there is not any clear evidence of general disturbance of health. In all cases, death has ensued through some coincident, not evidently associating disease, which has been aggravated by the conditions of the bones, only in so far as they may have diminished the range of breathing and the general muscular activity. Paget correctly interpreted the condition as being a chronic inflammatory process.

Considered as a rather rare condition, Clopton, in 1906, collected seventy-five cases from the literature. Higbee (3) and Ellis, in recording a case in the "Journal of Medical Research, 1911," state that the number has been increased to 158 cases, 33 of which were noted in this country. The relative increase during the past few years points to one of two things—that the disease is becoming more prevalent or is more often recognized as such.

Numerous names have been applied to this condition, which illustrates the views of different authors, according to their interpretation of the process:

Osteitis deformans (Paget).
Pseudo-rachitis senilis.
Osteo-malacia chronica deformans hypertrophica.
Ostéite ossifiante diffuse (Lanceraux).
Ostéolysis (Lobstein).
Hypertrophie spongieuse des os.
Craniosclérose.
Hyperostose généralisée.
Ostéite condensante.
Ostéomalacie hypertrophique benigne (Vincent).
Pseudorachitisme senile.
Osteomyelite fibreuse.
Sclérose osseuse hypertrophique.

In reference to the etiology, little is known. In spite of the many theories that have been advanced, there is no generally accepted etiological factor. The most important are of interest, in that they tend to show the wide divergence of opinion.

Lesions of the nervous system, hereditary lues, arterio-sclerosis, metabolic changes, and disturbance in secretion, particularly of the thyroparathyroid group, testicular, and suprarenal glands, have all been suggested as etiological factors.

Prince (4) suggests, that it is not inconceivable that as degeneration of the anterior cornua may cause myopathies without other symptoms; so lesions of other special cell groups may cause osteopathies without other spinal symptoms. Curcio (5) also suggests the possibility of a special trophic center in the gray matter of the spinal cord because the manifestations of the disease are usually symmetrical.

Syphilis: Some observers, particularly, Lannelongue (6), regard the disease as a manifestation of congenital syphilis or a paraluetic condition, asserting that improvement has been noticed following the administration of mixed treatment. Fournier (7) agrees with Lannelongue and mentions the case of two brothers one of whom presented a typical clinical picture of Paget's disease, the other showing marked evidence of congenital syphilis. He later speaks of the disease as a paraluetic condition, to be classed with tabes dorsalis, general paresis, etc. Von Recklinghausen (8) considers that the disease is not specific. Robin (9) endeavored to differentiate Paget's from the luetic osteitis by comparison of the chemical composition of the bones, but his results are of little value at present, as only one case was analyzed. Weber (10) says that the osteitis deformans of congenital syphilis and Paget's may be differentiated as follows: 1. The youthful age of the patient in cases of congenital syphilis; 2. The relative absence of pain, which is almost constant in Paget's; 3. The favorable results obtained in syphilitic cases with proper treatment; 4. In syphilitic cases the tibiae are most severely affected, in Paget's disease the femora; 5. The presence of irregular bosses in congenital syphilis; 6. There is no evidence of any tendency for malignant tumors to supervene in the bones of syphilitic cases, as there is in Paget's.

Heredity: Several instances are recorded in which members of the same family have been afflicted with this disease. Chauffard (11) mentions mother and daughter; Smith (12), a father and son; Berger (13), a mother and son. Paget, however, in reviewing 27 cases, remarked on the entire absence of any such history.

Occupation: Oettinger (14) and Agasse Lafont noted that several cases had occurred among those whose occupation, such as bleachers, tanners and hatters, subjected them to the action of certain acids. They considered it possible that the chemicals may have some action on the osseous structure

Arterio-Sclerosis: This condition is present so frequently that it may be considered almost constant. Not a few, particularly Béclère (15), have considered that the interference with nutrition, resultant upon the sclerosis of the nutrient arteries of the bones affected, may have some etiological bearing.

Heitz (16) and Hudelo speak of a woman 75 years of age, in which the skull was unchanged but whose femora and other bones presented lesions of Paget's disease. Arterio-sclerosis was not uniform. In this instance the arteries of the brain, skull and neck were normal, while those of the remaining viscera showed marked sclerosis, even to obliteration.

Interference with the function of the glands, and disturbance in the internal secretions has been considered. Our case is interesting in the entire absence of any change in the glands of internal secretion, so far as determined. The lesions of the nervous system described in cases of Paget's disease should be accepted with considerable reserve. We believe this because of the pronounced arterio-sclerosis almost constantly associated with the disease, and because of the senile changes so frequent in brain and spinal cord, in old age. On account of the frequent association of arterio-sclerosis with the disease, it seems reasonable to suppose that it may act as a causative factor.

It is of interest to note that Max Koch (17), before the German Pathological Society, demonstrated a case of Leontiasis ossium, and showed that the microscopical picture was similar to that occurring in Paget's disease. He therefore considers them identical.

We are unable to explain in detail how the vascular changes could so markedly alter the osseous tissues. That the process is evidently not always a generalized one leads us to suppose, that more consideration should be given to local disturbances, than to a general disturbance involving the osseous system as a whole.

Perhaps, as Higbee and Ellis state, accurate studies of the metabolism offer the most hopeful means of arriving at the proper solution of this question. Although we favor the idea that it is a local condition and not a systematic one, we realize that there must be some other caustive factor present, for the reason that so many cases of marked cerebral arterio-sclerosis show no changes in the osseous system.

Miscroscopic examination of sections from our case show as follows:

Occiput: Section taken from the occiput—the outer table is formed of a thin layer of dense bone; there is little differentiation in structure of the bone. The inner table is very thin; the marrow spaces are large and everywhere filled with connective tissue composed of a homogeneous stroma and connective tissue cells with small spindle-shaped nuclei. The vessels are large; there is no proliferation of their endothelial cells. In places the connective tissue is more fibrous in character; the osseous trabeculae are large; there is well developed concentric lamellar formation; the bone cells are small; there are few Howships lacunae and only in places are there osteoblasts. To be brief, the process may be described as an osteitis or osteomyelitis fibrosa; the trabeculae are large, well connected with each other, and there is everywhere evidence of a formative osteitis. The bone was extremely difficult to decalcify, and there is every evidence that there was no marked halistresis.

Tibia: Section taken from the cortex of the tibia, which was thickened as above mentioned, shows very dense bone.

Right Femur: The cortex is formed of rather dense bone with large vascular marrow spaces, showing an osteogenic connective tissue very similar to that found in the occiput. Only a few giant cells are seen. The periosteum is thickened and fibrous; the inner layer is cellular.

Left Femur: The section taken at a level corresponding to that of the right femur is normal.

Vertebrae: Shows normal lymphatic marrow, in places quite fatty.

Ribs: The marrow spaces are very wide; trabeculae long and narrow.

Clavicle: Shows no noteworthy changes.

The gross lesions and the microscopic sections indicate that there has been an extensive formative osteitis of the calvarium, correponding to what Von Recklinghausen has described as an osteitis fibrosa.

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CASE OF MULTIPLE MYELOMA.*

CHARLES NORRIS, M.D. AND B. MORGAN VANCE, M.D.

The organs came from a man 54 years of age, a window dresser by occupation, who entered the service of Dr. Draper at Bellevue Hospital on January 26th, and died October 20th of the present year. Seven months before admission he complained of stiffness and tenderness in the neck and ribs; the pain increased rapidly so that in the next two months he was forced to go to bed. The pain was worse on movement. A week before his death he developed pneumonia from which he died.

The body was poorly nourished, 176 cm. in height. The arms and legs show extreme emaciation; skin is pale and of fine texture. The lungs are everywhere congested and slightly oedematous; the upper right lobe is in a condition of red hepatization. The kidneys are large and congested. Spleen is somewhat small and fibrous. The other viscera show but little change from the normal. The sternum is thickened, soft and everywhere infiltrated with fusiform, swellings, the largest about 3 inches in length. The tumor infiltration in the ribs is very extensive. The clavicles are also infiltrated in places with tumor tissue. The cervical vertebrae are soft, are easily penetrated with the point of a knife, and there is a considerable amount of tumor tissue which has grown through the periostium of the vertebra forming a flat mass of yellowish white tissue in front of the upper cervical vertebrae. The basilar portion of the occipital bone is softened and can be readily cut away with a knife. The lower end of the right femur shows a focus which is dark red in color; otherwise the bone marrow is normal. The upper half of the left femur is extensively infiltrated with tumor tissue with large haemorrhagic-like areas. A few of the dorsal vertebrae examined show small tumor areas.

The microscopic examination of the viscera shows but slight changes besides those mentioned above. There are, however, recent areas of focal necrosis of small size in the spleen and the muscle of the anterior abdominal wall shows a well marked hyaline degeneration. Examination of the sections taken from various portions of the bone infiltrated with tumor growth shows that the tumor is made up of two kinds of cells—a large cell with protoplasm in which no granulations can be made out, with a large nucleus occasionally eccentrically placed with a definite nuclear membrane and with scanty chromatin network. The other cell is smaller in size with a dense, small, deeply staining nucleus and with a markedly eosinophilic protoplasm. The tumor cells are free and there is only a very scanty amount of reticulum. It is probable that these latter cells were erythroblasts and that the two types of cells were derived from cells analogous to the primitive blood cells of Maximow. Smears made directly from the fresh tumor and stained show no granulations. Examination of the tumor cells in the hanging drop likewise show no granulations. During life the urine contained very large amounts of Bence Jones protein. A full description of the clinical history and the microscopic findings and conclusions will be published elsewhere.

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A LARGE TUMOR OF THE LIVER, ASSOCIATED WITH A TUMOR OF THE OESOPHAGUS.*

CHARLES NORRIS, M.D.

The liver and oesophagus which I show you came from a man 73 years old, with very little clinical history, except that he had been, up to middle age, a severe drinker, and three months before death he had epigastric pain and lost weight and became very weak.

At autopsy there was found a very large liver which practically occupied the right half of the upper quadrants of the abdomen. A clinical diagnosis of primary carcinoma of the liver had been made and, before I came across the tumor of the oesophagus, it seemed to me that that was a likely diagnosis, the tumor resembling some of the cases of primary carcinoma of the liver that I have seen. An interesting observation in this case is that, although the metastatic growth in the liver was so extensive, there were no metastases elsewhere with the exception possibly of one, say only 3 or 4 mm. in length by one mm. in breadth on the pleura of the left lower lobe of the lung. No metastatic nodules were found in the other organs on microscopic examination.

The tumor in the oesophagus is situated at the bifurcation of the trachea, measuring 7 cm. in length by 3 cm. in breadth. About 1 cm. of the wall of the oesophagus is free from growth. There is no stenosis of the oesophagus. The portion of the trachea beneath the tumor of the oesophagus is uninvolved, the mucous membrane being normal in the gross.

The growth in the liver and oesophagus were identical as to their histology. I am unable to make up my mind, for the present, whether to call the tumor an epithelioma or a sarcoma.

EXTENSIVE IRREGULAR NECROSIS OF THE LIVER IN AN INFANT.*

C. W. FIELD, M. D. AND A. M. PAPPENHEIMER, M. D.

The liver which we wish to demonstrate was obtained at autopsy from an eighteen-day-old infant. The baby was one of twins born at eight months. Labor was simple, the puerperium uncomplicated.

The infant was well until the sixth day, when it refused the breast, and had to be fed with a medicine dropper. On the following day, it became slightly icteric and oedema developed about the feet. The oedema during the following days decreased; then reappeared, extending gradually to above the navel. There was also transient oedema of the face. The icterus diminshed gradually, and disappeared entirely before death, but spasmodic twitchings developed and the child passed into coma, in which it died on the twelfth day of its illness.

The twin child was perfectly well until the seventh day. Then it too suddenly became unable to nurse, and developed oedema, and progressive icterus. A diminution in the amount of urine was noted, but no examination was made. The child died after four days and no autopsy was obtained.

The autopsy on the first child, which was held eighteen hours postmortem, showed besides the lesions in the liver, double broncho-pneumonia and suppurative inflammation of the left mastoid. The appea ance of the liver was striking. The organ was about normal in size, the surface was smooth, the edge sharp, the consistence somewhat firmer than normal. The whole organ,

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both on the surface and on the cut section, showed a peculiar mottling—irregular areas of a greenish gray, slightly translucent appearance, alternating with reddish and yellowish red areas. The grayish areas, which were found subsequently to represent intact liver tissue, were slightly more prominent above the surface than the reddish portions.

They were sharply circumscribed, and on close inspection it could be seen that they were in relation to, and formed irregular mantles about, the larger portal spaces.

The mottled appearance observed in the gross is even more striking and distinct in the stained sections. Here one may discern sharply demarcated, irregular islands staining more deeply than the surrounding tissue and corresponding, under the microscope, to areas of well preserved liver cells. By far the greater portion of the liver, however, is composed of liver cells which have undergone regressive changes.

In sections stained in hematoxylon-eosin, the degenerated areas show no trace of lobular structure. The individual cells no longer show a columnar arrangement but are irregularly disposed and separated one from another. The degenerated cells are distinctly smaller than the normal cells. The protoplasmic outline is often indefinite. The cytoplasm of the affected cells does not take up the hematoxylon but shows a strong affinity for the eosin. With the immersion, the protoplasm has a foamy, vacuolated structure and in frozen fixed sections the cells are found filled with smaller and larger droplets staining bright orange red, with Sudan III. Nowhere, however, are there seen larger globules completely filling the cells and compressing the nucleus.

When treated by the Ciaccio method for the demonstration of the more complex lipoids, the degenerated cells show numerous droplets staining a dirty brownish yellow with Sudan III. Free flat globules are also found in small numbers within the capillaries. The nuclei are shrunken in comparison with that of the normal cells, approximately not more than half the size. They are circular in outline and stain densely and diffusely. Occasionally they are in the form of irregular, pycnotic fragments lying at the periphery of the cells; ocassionally also signet and crescent forms are seen. Some of the cells appear to have lost their nucleus altogether, although this may be due to the fact that the nucleus lies without the plane of the section.

The islands of unaffected liver cells form irregular mantles about the larger portal spaces and are also preserved in places as a thin layer just beneath the serosa. All the portal spaces, however, are not surrounded by normal liver cells. In general the islands of normal cells preserve their characteristic lobular structure and radial arrangement about the central vein. The individual cells are approximately of normal size, showing no hypertrophy when compared with cells from the normal liver of an infant of approximately the same age.

No fat globules or glycogen are present, but in the Ciaccio preparation many of the normal cells show a diffuse reddish brown discoloration. The capillaries in the normal areas are distended with well preserved blood cells. Here and there are found small groups of cells which under the immersion can be identified as normoblasts and megaloblasts. Large cells with a vesicular nucleus and somewhat more basophilic cells, probably representing precusors of the megaloblasts, are also found. In a few of the megaloblasts, karyokinetic figures were seen. These erythoblastic centers, which were studied in Giemsa-stained sections, are seen not only in the capillaries of the normal liver tissue but also in the degenerated areas. Moreover, nucleated red cells of all types are present in unusual numbers throughout the liver. There is, therefore, an active hemopoesis, apparently unaffected by the degeneration of the parenchymal elements.

There is very little fibrosis or evidence of active connective tissue hyperplasia. Some of the smaller vessels in the periphery of the lobules are surrounded by apparently actively growing fibroblasts, and in the neighborhood of the large portal trunks the connective tissue of Glisson's capsule is somewhat more abundant than normal. With Bielschowsky's stain no increase in the intralobular connective tissue is apparent either in the normal or the degenerated regions.

There is in many places evidence of regenerative activity on the part of the smaller bile ducts. From the periphery of the lobules we see small ingrowths of cells with dark staining protoplasm containing various numbers of irregularly-sized vesicular nuclei, some of which are in active karyokinesis. Three mitotic figures were seen in a single mass of cells. The direct connection of these cell masses with the bile ducts in the portal spaces and at the periphery of the lobules can in some places be demonstrated in serial sections. The bile ducts give off lateral off-shoots which soon lose their lumen, branch, and penetrate the periphery of the lobules for a short distance. The cells are no longer cylindrical but irregularly polygonal, and tend to lie in double rows, though the terminations of the sprouts may be three or four cells in thickness.

Owing to the methods of fixation employed, no bile can be demonstrated in these new formed cells; neither do they contain fat in appreciable amount, or glycogen. The efforts at regeneration displayed by the intact liver cells are negligible. A rare mitosis is found, but there is no reduplication of the cells of the liver cords, no loss of the radial arrangement, no formation of large multinuclear cells and no ingrowth of new formed cells into the degenerated lobules. This is probably due to the short course of the disease, affording no time for the development of the adenomatous hyperplasia described by Meder (1), Merchand (2), Stroebe (3), MacCullum (4) and others in long standing cases of liver atrophy. Regeneration, in this case, therefore, is practically limited to the solid sprouts derived from the interlobular bile passages. Active and conspicuous as is this proliferation, it is not probable that it has in this case led to the regeneration of functional liver cells. The terminal solid buds resemble liver cells in the appearance of their nuclei, but the cells do not assume a characteristic polygonal shape of normal liver cells, nor do they enter into a characteristic arrangement with the persistent capillaries. Furthermore, regressive changes may occasionally be noted in the nuclei of the terminal cells of the sprouts, showing that some at least of the new formed cells are short lived.

Very striking and significant is the absence of acute inflammatory changes. There is no accumulation of small, round cells in the portal spaces, no infiltration of polynuclears. Neither can there be observed the phagocytosis of necrotic cell detritus by the desquamated endothelial cells of the capillaries as described in cases of focal necrosis by Mallory. These features are opposed to the idea of a direct bacterial causation for the necrosis.

As in so many of the reported cases of acute atrophy of the liver, we are quite in the dark as to the real cause of the lesion. The finding of slight cirrhotic changes in the pancreas, together with rudimentary islands of Langerhans (some of which apparently show a direct connection with the pancreatic ducts) suggested syphilis as a possible factor. Syphilis has been brought into relation with acute yellow atrophy by a number of observers. Meder in 1895 was able to collect twenty such cases.

In the case here reported, no spirochetae were found in Levaditi sections from all the principal organs. This fact, together with the absence of other

characteristic syphilitic lesions, seems sufficient to exclude syphilis as a positive causative factor.

Since attention has been called to necrotic lesions in the liver, occurring as a sequel to chloroform narcosis, it might be suspected that chloroform administered to the mother during labor might account for the lesion. We have, however, ascertained that no chloroform was used in this case. No cultures were made at the time of autopsy, so that we cannot say whether or not the liver lesion is a manifestation of a general bacterial infection. The absence of micro-organisms in the necrotic areas in sections stained by Gram, methylene-blue and Giemsa, as well as the lack of inflammatory reactive changes, seem against this possibility. The lesion represents evidently what we are accustomed to call a toxic necrosis; whether due to bacterial or metabolic toxins it is not possible to say.

Our study of the literature is still incomplete, but we have thus far been able to find but one reported case of extensive necrosis of the liver occurring in young infants. This is the case published by Aufrecht (5) in 1896. The mother was a girl 19 years of age, primipara. The labor was normal. After twenty-four hours there was noticed in the child swelling about the feet, which on the second day extended above the symphysis. The swelling is described as hard, doughy and could be pitted on pressure. On the second day, also, there developed slight general icterus; the urine contained a small amount of albumin and "leucin spheres." On the third day the oedema extended to the navel and punctiform hemorrhages appeared on the left leg. The icterus increased. Death took place on the sixth day. At the autopsy, sixteen hours after death, aside from scattered petechial hemorrhages in the serous membranes, the lesions were confined to the liver. The organ is described as weighing 100 g., was firm, bluish black in color and showed no acinal markings. Microscopically there was complete loss of the lobular structure. The nuclei were shrunken and stained poorly. The cells were vacuolated. There was extensive hemorrhagic extravasation. Many bacilli were found in the capillaries.

Although the report of this case is somewhat incomplete, there seems to be a resemblance in the clinical history as well as the pathological findings.

Todt (6) in 1903 collected from the literature thirty-two cases of acute yellow atrophy in infants and young children. None of these, with the exception of the case above quoted, occurred in the new-born; almost all of them were similar clinically and pathologically to cases occurring in adult life. In conclusion we wish to call attention to the resemblance between the lesions in the liver of our case, and those produced in dogs by Pearce (7) by injection of large doses of hemolytic serum. As is well-known, he attributed the necrosis in his experimental lesions, to "obstructive congestion of the capillaries and the smaller branches of the portal vein by fused masses of red cells." In our case, there was absolutely no histological evidence of such a condition, and we have no basis for considering the lesion as the result of infarction.

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DEMONSTRATION OF SPECIMEN OF MEGACOLON, OR HIRCHSPRUNG'S DISEASE.*

CHARLES NORRIS, M.D.

The specimen came from a male child 18 months of age, who had been subject to constipation since the age of three weeks; for the past year had been treated at dispensaries for constipation. There were no natural movements during his week's stay in the hospital. The abdomen was greatly distended, the abdominal wall being very thin, due to extreme stretching. On cutting through the abdominal wall, the entire upper portion of the abdomen was occupied by a greatly distended transverse colon and coecum. The dilatation ceased abruptly at the splenic flexure, the descending colon and sigmoid being of normal appearance and dimensions. The dilatation commenced in the coecum at the ileo-coecal valve, the small intestines being normal. The dilatation is greatest at the middle portion of the transverse colon, the gut being in this situation 21 cm. in diameter. Wall of the dilated gut is thickened, its mucous membrane is everywhere smooth, showing in a few places patchy, mucosal hemorrhage. The gut contained a large amount of extremely frothy material which was light colored and greasy. At the splenic flexure there was a valve-like fold at the junction of the distended and undistended portions of the colon. It seems probable that the partial valve-like closure at the splenic flexure had led slowly to the distention and hypertrophy of the gut. Sections taken from the distended gut show marked hypertrophy of the muscular coats, mucous membrane being normal except for the hemorrhagic spots referred to above. The other organs showed no lesions of interest. The case is an unusual one for the reason that the sigmoid flexure was not involved.

DISSECTING ANEURISM OF THE AORTA.*

CYRUS W. FIELD, M.D.

The specimen came from a case of a colored man, 68 years of age, who entered Bellevue Hospital in the service of Dr. Alexander Lambert and died with a diagnosis of broncho-pneumonia and probable malignant endocarditis. The interesting feature of the case relates to the aorta.

On opening what was first taken to be the aorta, it was found that one entered a large arterial trunk lying to the right of the true aorta, and running parallel with it to its bifurcation. It was thought at first that one might be dealing with a case of double aorta; but further dissection showed that it was a dissecting aneurism. At a point in the aorta 2 cm. below the right subclavian, there is a circular opening 2 cm. in diameter in the aortic wall, leading into an aneurysmal tube, the internal surface of which is perfectly smooth and glistening, and which has the appearance of a large arterial trunk. Passing downward, the tube divides and follows both common iliacs, and the external iliacs beyond the inguinal canal. The right renal artery is given off from the true aorta; as it passes through the aneurysmal sac a portion of its wall has been eroded, so that there is an opening from the sac into this renal artery. The right kidney, therefore, derives its blood from both the circulation in the aneurysmal sac and the circulation in the aorta. That there was a free circulation within the aneurysmal system is shown by the absence of clot either in the main trunk of the aneurysmal sac or in any of its branches.

During life the patient gave no symptoms referable to such condition. It is interesting to note that in this case the Wasserman reaction was negative.

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THE OCCURRENCE OF A POSITIVE WASSERMANN REACTION IN CASES OF LEAD-POISONING.*

CYRUS W. FIELD, M.D.

During the past year the routine Wassermann examinations in Bellevue Hospital have shown a number of cases of lead-poisoning giving a positive reaction; and for this reason we desire to call attention to the fact though its occurrence has been noted incidentally before.

We have had twelve cases of lead-poisoning in which the blood had been sent into the laboratory for a Wassermann test (1), and of these twelve, eight were positive and four negative. Of these eight positive cases, only one gave a history of ever having contracted syphilis and in that case it was thirty years previously and the patient claimed never to have had any secondaries or tertiary lesions. His wife had had one miscarriage. Another of the eight patients had a very suggestive specific history. A woman, aged 46, a domestic, gave a history of having had one child and at least five miscarriages, but denied all venereal history. She was evidently very susceptible to lead-poisoning, for she had an attack five years previously (after painting some furniture, according to her own statement), suffering at that time from marked wrist-drop. No recent history of exposure to lead could be made out, but she presented at this time a slight double wrist-drop abdominal pain, lead line on the lower gums and her blood showed marked granular basophilia of the erythrocytes.

In one other case no history could be obtained, as the patient entered the hospital in a semi-comatose condition and died shortly after. He was a solder-caster by trade and died of cerebral hemorrhage. His red cells showed marked granular basophilia and the anatomic diagnosis at autopsy was as follows: cerebral hemorrhage, right lenticular artery, with rupture into lateral ventricles; lobular pneumonia (right and left); serofibrinous pleuritis; healed apical tuberculosis; hypertrophy left ventricle; chronic valvulitis, aortic and mitral; chronic aortitis; chronic interstitial nephritis; chronic interstitial splenitis; nutmeg liver; otitis meda, right, purulent.

In the other five cases there are no signs or symptoms in any way referable to a specific lesion, all being typical cases of lead-poisoning. All the patients denied any venereal disease, but all five showed a positive Wassermann reaction. None of these came to autopsy, all having been discharged from the hospital improved after treatment with potassium iodid, strychnin and catharsis.

The four negative cases were all cases of chronic lead-poisoning occurring in painters. Syphilitic infection was admitted by one of these with secondary manifestations forty years ago, but he had since had no symptoms referable to syphilis. None of them could be called alcoholic in any way. One of these patients died and came to autopsy, the anatomic diagnosis being: lobular pneumonia, right upper lobe; tuberculosis pleuritis, left; chronic pulmonary tuberculosis, left upper; chronic interstitial nephritis; fatty and cirrhotic liver; chronic interestitial pancreatitis; intestitial thyroiditis; chronic aortitis; chronic gastritis; chronic pharyngitis and laryngitis with acute edema; calculi of seminal vesicles; pial edema.

In the literature there are a few cases of lead-poisoning which have been reported with positive Wassermanns, but we cannot find that the condition has been considered as a possible source of error in interpreting the Wassermann reaction. Of course, it would be impossible to say, from the few cases here

*From the Pathological Laboratory Bellevue Hospital, New York City.
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reported, that the positive reaction was brought about by the lead-poisoning, for it may be that cases of lead-poisoning are more apt to give a positive Wassermann with an old syphilitic lesion than are others; and owing to the fact that not all cases of lead-poisoning that entered the hospital were examined for the Wassermann reaction, it would be improper to consider our series as showing the correct proportion of cases of lead-poisoning giving positive Wassermanns. It will probably be found to be lower when more cases are examined.

Another point which would be well worth observing, but which we have been unable to do in any of our cases so far, is to see what effect an injection of salvarsan or mercury, or both combined, might have on the Wassermann reaction.

In conclusion, we cannot at the present time accept a positive Wassermann reaction in a case of lead-poisoning as proving a specific infection, and, on the other hand, we cannot ignore its presence. Until many more observations are made and much experimental work has been done, it will be best to simply keep this fact in mind in interpreting positive reactions in persons exposed to lead.

1. The technic of the Wassermann reaction used in this series is as follows: A 5 per cent. solution of sheep red cells and amboceptor obtained from rabbits and as antigen, the acetone-insoluble fraction of lipoids obtained by extracting syphilitic fetal livers with alcohol, taking up in ether and then precipitating with the acetone. This is redissolved again in ether and reprecipitated. The precipitate is then dissolved in Merck's methyl alcohol. The amount of this used is 0.01 c.c.. The patient's serum is tested to see whether it contains any natural amboceptor for sheep-cells and this is taken into consideration when the hemolysin is added. It is needless to say that all tests are controlled. The quantities are one-half those used in the original Wassermann technic.

A COMPARATIVE STUDY OF THE WASSERMANN AND WEIL REACTIONS IN SYPHILIS.*

Cyrus W. Field, M.D.

In 1906 in the Journal of Infectious Diseases, Weil described the occurrence of an increase in the resistance of red cells to cobra venom lysin in patients suffering from syphilis. A little later Schwartz reported on 600 cases in which he compared the Wassermann and the Weil reactions. This year Stone, of Toledo, reported another series; and Kuschucoff from Wassermann's laboratory has also reported a series.

Last spring we began a routine Weil test along with our routine Wassermann. We found that the Weil reaction was positive in 76% of primary syphilis, in 75% of secondary, in 73% of tertiary and 40% in questionable and negative cases and 50% in cases suffering from tumors, whereas the Wassermann reaction in the same series showed 70% positive in primary syphilis, 87% secondary, 75% tertiary and 30% in the questionables and negatives. These figures are almost alike except that the Wassermann reaction gives a higher percentage in the secondary cases.

It was found that there were a few cases in which we obtained a positive Weil reaction in which there was absolutely no suggestion of syphilis or malignancy and we determined to run a series on absolutely normal individuals and so obtained the blood from eighteen of the internes of Bellevue Hospital for the Weil test. In sixteen of these cases enough was obtained to control it with the Wassermann. All sixteen were negative with the Wassermann. The two on whom there was no Wassermann test were both negative with the Weil reaction. In the other sixteen cases the Weil reaction was strongly positive in one, and a doubtful positive in two, negative in all the rest. None of these three individuals have any history, signs or symptoms which would indicate a specific lesion in either their personal or family history.

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From our work with the Weil reaction it would seem to be of value in known cases of syphilis undergoing treatment and possibly where the Wassermann reaction could not be readily undertaken. The test itself is of very great theoretical interest as it shows that there is a distinct difference in the chemical constitution of the erythrocytes in normal individuals and in those suffering from syphilis and new growth. We do not think that one strong positive in eighteen is a fair number of positives from non-syphilitic individuals as it will give a high percentage, which would not be found if the number of cases were greater, though we do think that it will be found that the Weil test will give a larger proportion of positive results in non-luetic conditions than does the Wassermann.

EDEMA OF THE PIA-ARACHNOID—ITS NATURE, SIGNIFICANCE, RELATIONSHIP TO AND ASSOCIATION WITH DISEASE PROCESSES.*

CHARLES K. STILLMAN, M.D.

The following article is a study of the nature and significance of fluid accumulations within the subarachnoid space, based principally on post-mortem observations and mechanical considerations.

For the sake of brevity it may be stated at the outset that the terminology employed to designate the various conditions of the brain and its membranes associated with excessive fluid accumulations has been confused by the use of many names, employed without due consideration of the etiological or mechanical factors involved. It is therefore necessary to describe the various anatomical findings to which these different terms have been applied.

1. **Pial Edema; Edema of the Pia Mater; Edema of the Pia Arachnoid; Cerebral Edema; Wet-Brain.**—These terms have been employed indefinitely to designate conditions accompanied by excess of fluid in the pia-arachnoid. The brain on cut section may or may not appear to be moist; the ventricles may or may not be "dilated." The volume of the brain is not increased, on the contrary it often has given the impression of having been compressed. It is this picture which is generally associated with and attributed to chronic alcoholism, and which in this country has often been referred to as wet-brain.

2. **Cerebral Edema; Edema of the Brain.**—Swelling of the brain with increase in its volume. The pia-arachnoid may or may not have contained an excess of fluid; usually the convolutions are described as flattened, owing, as supposed, to pressure of the voluminous brain against the skull. These brains are then described as being moist, the condition being usually attributed to transudation into the brain substance of serum from the blood-vessels.

3. **Serous Meningitis.**—Described usually as a serous or exudative inflammation of the soft meninges, with exudate into the subarachnoid space and its prolongations, with or without gross thickenings of the meninges. The ventricles may or may not be dilated and the brain is or is not secondarily involved, the seat of an edematous or inflammatory process. Certain writers in this country, notably Dana, have regarded "alcoholic wet-brain" and serous meningitis as synonymous.

4. **Hydrops ex Vacuo.**—This term has been used to describe the accumulation of fluid following the shrinkage of the brain from old age or from various pathological causes, to be mentioned later.

*From the Pathological Laboratories of Bellevue Hospital, Dr. Charles Norris, Director.
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5. **Chronic External Hydrocephalus.**—This term has been used to describe those appearances already referred to above; by some authors, also, to describe accumulations of fluid in the subdural space and even those found between the calvarium and the dura mater (1).

The above summary, derived from a critical survey of the literature, shows that these various terms, although in themselves perfectly clear and descriptive, have been used indiscriminately to designate different anatomical conditions without any consideration of the underlying pathological processes in which they had their origin.

Although the older writers did not formulate any noteworthy theories as to the causation of excessive accumulations of fluid in the brain (2), they nevertheless displayed considerable interest in its supposed mechanical effects. Thus we find in Watson's Lectures (3) (1845), the following rather quaint description of fluid collections in the pia-arachnoid in connection with uremic coma.

"Now when death had thus taken place in the way of coma and the case had been complicated with anasarca, and serous liquid is found accumulated in unnatural measure in the cerebral ventricles, and in the tissue of the pia mater, it seems reasonable to ascribe the coma to the presence and the pressure of that liquid. The dropsy has extended to the brain. And this view of the matter is strengthened by the connection which may sometimes be noticed between the accession of the coma and the visible increase of the dropsy in other parts of the body. My own experience accords entirely with that of Dr. Christison as expressed in the following statement: "If the dropsical fluid be allowed greatly to accumulate, drowsiness, the first symptom of the affection of the head, very soon makes its appearance in the generality of cases and it will speedily pass to fatal coma if not controlled, but the removal of the dropsy will usually remove the drowsiness." To many cases, however, this explanation will not apply, there being no morbid collection of water within the skull, nor any other appreciable change there; nor perhaps, any dropsy elsewhere."

The fluid collections described by Watson were doubtless that very common form of pial effusion with which this paper particularly has to do.

In 1861, Traube (4) opened up a new vista in the field of intracranial mechanics by stating that the brain is often swollen and edematous in certain cases of uremia. Since Traube (5) may thus be regarded as the founder of a new school, a somewhat detail d consideration of his paper is imperative. In discussing uremia he says: "In all cases observed by me in which a careful examination of the cranial contents has been made after death, there was observed with a well-marked anemia (Blut-Armuth) of the brain, a more or less considerable edematous swelling of the same (as shown by flattening of the brain convolutions and dryness of the arachnoid, with moist glistening of the cut section)" (6). Further on he writes: "In some of these cases the fresh petechiae (Blut-Ergüsse) were distributed in the form of fine sand up to hempseed foci through the large and small brain and pons due," as he states, "to increased arterial pressure" (7). He lays especial stress upon two factors in the causation of brain edema in uremia; first, the hydremic condition of the blood, due to loss of albumin through the urine and through gastro-intestinal catarrh, and second, to the increased tension in the aortic system from hypertrophy of the left ventricle (5). He goes on to say:

"If through any accidental cause there occurs a sudden increase of blood-pressure, or if there is a sudden diminution in the thickness of the blood-serum, there transudes a serous fluid through the walls of the small arteries into the brain substance, and thus brain edema ensues. The fluid elements of the blood transude under the mean pressure in the aortic system. Since this pressure is

greater than that in the capillaries and veins, so these vessels must finally be compressed. The necessary consequence of a brain edema arising from such a cause is an anemia of the brain substance. The transudation, as will be readily understood, resists up to the moment when the tension of the edematous brain parts equals the mean tension of the aortic system."

Traube's explanation of the causes bringing about transudation is not generally accepted. He cites a case of cerebral edema in an epileptic who at autopsy was found to have a cysto-sarcoma of the brain. There is reason to believe that from this case he derived the idea on which he founded his theory as to the causation of the attacks of coma and convulsions occurring in cases of uremia and of chronic lead-poisoning.

Several years after Traube's paper appeared, Billroth (8) (1869) observed anatomical changes in the brains of postoperative subjects somewhat similar to those described by Traube.

Niemeyer (9) (1877) and Huguenin (10) (1878) reconsidered the problem chiefly in connection with the phenomena of uremia.

Bergmann (11) (1880) described an edematous condition in connection with injuries of the brain, while Dean (12) produced local edema experimentally by placing a glass disc between the brain and the dura.

Phelps (13) (1897) believes that edema of the brain may follow on contusion and W. N. Bullard (14) (1895) states that edema of the brain results from concussion. The latter writes:

"Brain swelling undoubtedly occurs in certain cases of apoplexy, and in a chronic form in many intracranial diseases. This, or something analogous to it, the so-called acute edema of the brain, is the immediate cause of death in cases of acute alcoholism, of sunstroke and perhaps (in its chronic form) in uremia."

Walton and Brooks (16) (1897) have also dwelt on the subject of edema of the brain and its membrane from a surgical standpoint. Walton in his subsequent paper (17) has considerably modified his previous conceptions.

W. B. Cannon (18) in an article published in 1901, presented certain striking and original conclusions on the causation of cerebral edema following trauma, which will be later referred to, while at about the same time Mott (19) succeeded in producing experimental edema of the brain in dogs by ligation of arteries, and Osler (20) (1901), following Traube, described cerebral edema particularly in connection with uremic coma.

The subject of cerebral edema has aroused great attention among physiologists only within a comparatively recent period. Wilson (21) (1904) studied the condition in connection with uremic and eclamptic seizures. B. Bramwell (22) (1906) mentioned cerebral edema as a possible cause of uremic attacks. The following year A. E. Russell (23) (1907) introduced his thesis in the following terms: "The purpose of this paper is to put forward the proposition that there is evidence to show that the cerebral manifestations of uremia are dependent on cerebral anemia produced by an increase in intracranial tension resulting from cerebral edema." Following Russell, Cushing and Bordly (24) (1908) published a paper based chiefly upon the observations and finding ante- and post-mortem in a case of uremia in which decompression was performed, as a result of which he concludes: "In regard to uremia, therefore, to be conservative we may at least say that the symptoms are elicited by edema resulting from some toxic agency, and are not, as is commonly supposed, due to the direct effect on the cerebral tissues of the toxic agent alone" (25).

Russell (26) has very recently (1909) presented additional evidence supporting the hypothesis that the presence of edema of the brain is an important

factor in the production of uremic phenomena. He says, speaking of Cushing's decompression case (mentioned above) and other recent data:

"The foregoing facts indicate that in uremia a state of increased intracranial tension is present, and that relief of pressure by lumbar puncture or decompression causes a marked alleviation of the symptoms. Traube's view that cerebral edema (which would produce the rise in the intracranial tension) produces anemia of the brain is strongly supported by the above facts and especially by the striking case of decompression."

Russell does not deny a concomitant toxic action on the brain but thinks it possible that status epilepticus may be due to an anemia of the brain following on a prolonged increase of intracranial pressure. There is, as has been shown, a very strong tendency among the members of the Traube school to assume that the rise in intracranial tension is due to cerebral edema. As a matter of fact cerebral edema is a very inconsistent phenomenon in uremia, as has been noted not only by Russell but by Bramwell, and by a large number of authors who are not identified with the Traube school, namely, Huguenin, John Rose Bradford (28), Senator (29) and Riesman (30).

In this connection the observations of Kolisko (31) on swollen and edematous brains, are most interesting. This observer has described a type of brain in which the brain is swollen and wet, the ventricles reduced in size and the fluid of the subarachnoid space diminished or absent, the convolutions flattened, associated with dural hernias, and marked *impressiones digitates*—the latter being due to a compression atrophy of the inner table of the skull described as following upon a premature ossification of the cranial sutures.

The clinical phenomena in these cases have not appeared to bear much resemblance to those recorded by the Traube school.

The term "serous meningitis," the etiological and anatomical status of which is intimately involved in any consideration of sub-arachnoid effusions, is of comparatively recent introduction, although its employment antedates the bacterial era of medicine.

Since the development of our subject depends chiefly on anatomical rather than general clinical considerations, we shall regard serous meningitis chiefly from the anatomical and mechanical point of view, and limit our review of it.

Among earlier writers it was usual to ascribe all cases of internal and external hydrocephalus to tuberculous meningitis, although certain observers, notable among whom were Dietl (32), Rokitansky (33), Wunderlich (34) and Leubuscher (35) did not agree with this opinion.

In 1869 Billroth (35) distinguished a form of acute hydrocephalus of supposed non-tuberculous origin to which he applied the name of meningitis serosa. But it was not until Eichorst (36) (1887) published his observations that much emphasis was laid on the idea that a simple serous inflammation was the pathological process in many of these cases. Eichorst believed that the process was due to a mild infection and remained serous throughout, while Gowers (37) (1892) described the process as a meningitis simplex as distinguished from a meningitis purulenta. He was followed shortly by Quincke (68) who elaborated a definite symptomatology for this disease.

The literature of serous meningitis has become so enormous that it is out of the question to attempt to review it, especially since Boenninghaus (39) has given a comprehensive summary of the subject up to 1897 and Hafslauer (40) has subsequently reviewed it up to 1906.

The subject did not receive much attention in the United States until Dana (41) (1897) published his article entitled "Acute Serous Meningitis (Alcoholic Meningitis, Wet-Brain)." Since then, articles, monographs or reports of cases

bearing on the subject have appeared in this country by Lambert (42), Smith (43), West (44), Gradle (45), Stillman (46), Fischer (47), Diller (48), Collins (49), Tod (50), Spiller (51) and Stein (52), and abroad, either coincidently with or since Hasslauer's summary by Verhoogen (53), Hasslauer (54), Blau (55), Avellis (56), Riebold (57), Thiemich (58), Paradis (59), Axhausen (60) and L. Williams (61). A notable phase in the development of this subject of serous meningitis in the last few years has been the interest and attention which it has received from otologists.

The etiology is so much in dispute and the descriptions of this process by various writers are so divergent, not only from the clinical but also from the anatomical standpoint, that it is impossible to define precisely what is meant by this term This state of affairs makes it also impracticable to dwell on the mechanical problems involved. It is clearly out of the question to analyze the hundred or more articles which have appeared with this name for a subject.

In general, however, it may be said that an external and an internal form of serous meningitis are recognized and that these may be acute or chronic. In the acute external form the cortical pia arachnoid is described as being inflamed and infiltrated with round cells or leukocytes with serous exudation into the subarachnoid space and its prolongations. Sometimes the adjacent brain tissue is inflamed and edematous. When chronic, the pia may or may not be described as thickened. In the internal form there is transudation into the ventricles with dilatation. Quincke and others (62) have described the fluid as clear. Boenninghaus (63) believes that acute idiopathic hydrocephalus follows acute serous meningitis, while Thiemich (64) apparently considers that the "so-called acute hydrocephalus of childhood" and "ventricular serous meningitis" are identical.

Both Boenninghaus and Quincke have noted that non-inflammatory collections of fluid in the pia-arachnoid space are easy to mistake for serous meningitis. These pial effusions were discussed by Huguenin many years previously. He noted their association with certain atrophic states of the brain. I consider that knowledge of the subject has not greatly advanced since Huguenin's time.

Of late years much attention has been given to the abnormally large collections of fluid so often seen beneath the arachnoid membrane in subjects that have died of chronic alcoholism. It was primarily with the purpose of shedding some light on this subject that my paper was begun.

It became clear to me that post-mortem examinations of the brain alone would never throw sufficient light on the causation of these accumulations of fluid. I determined therefore to approach the subject by the following methods:

1. By means of an analysis of a series of cases, to show the frequency of association of pial edema with various diseases and to determine the influence of other factors such as age and nutrition.

2. By a consideration of the physical phenomena underlying the accumulation of fluid in the cranial cavity.

3. To determine whether there were any chemical differences in the cerebrospinal fluids in these various conditions that might indicate their etiological relation.

4. By the estimation and comparison of brain weights or volumes and cranial capacities in a series of cases.

With this brief introduction I now proceed to describe my observations.

Part I: Analysis of Cases According to Age, Nutrition and Disease.

Pial edema is a very common condition at autopsy, for out of a total of the 665 cases which form the basis of this report, it was recorded 375 times, a percentage of 56.4.

The cases with pial edema (375) have been placed in one group, those without pial edema in another.

Age.

An examination of the appended table shows: First, a relative frequency of pial effusion in infants from 4 months to 5 years of age (65).

Second, an entire absence of this condition between the ages of 6 and 15 years, followed by a rapid increase that culminates between the thirty-fifth and fortieth years with later a more gradual increase that reaches the maximum at from 80 to 85 years (old age).

The youngest subject with pial edema was 4 months old. When it is observed that in this series there were fully thirty-one cases of children, between birth and the fourth month, with dry pias, this initial date may not be without some significance (66). The absence of pial edema in nine subjects between 6 and 15 is also significant (67).

In the examination of fourteen cadavers between the ages of 16 and 20 years, pial edema was found twice, or in 14.2 per cent.

In an examination of thirty-four cadavers between the ages of 21 and 25, 26.4 per cent. presented pial edema. Of the nine, five showed a slight degree, three a moderate degree including one case of alcoholism, and one a marked degree (a case of cerebral syphilis).

In forty-five subjects between the ages of 26 and 30 years, 51.1 per cent., or twenty-three presented pial edema; five slight, nine moderate (including two alcoholics) and nine marked cases. It is important to note that in the nine marked cases, six subjects had lesions usually accompanied by brain shrinkage.

In sixty-four cases between the ages of 31 and 35 years, forty cases showed pial edema, twenty moderate, twelve marked, and one extreme (including eight

Fig. 1.—Chart, compiled from 604 cases, showing relative frequency of pial edema at various stages.

alcoholics and one case of insanity). The cause of death in the "extreme" case could not be determined.

In sixty-three subjects between the ages of 41 and 45 years, 61.9 per cent., or forty-one, showed pial edema; twenty-one of moderate, eleven of marked and two of extreme grade, including in all six alcoholics.

In fifty-six subjects between the ages of 46 and 50 years, 58.9 per cent., or thirty-three, showed pial edema; twelve of moderate and eighteen of marked grade, including four alcoholics.

In forty-six subjects between the ages of 51 and 55 years, 71.7 per cent., or thirty-three, showed pial edema; thirteen moderate, seventeen marked, one extreme (pulmonary tuberculosis). This group included two cases of chronic alcoholism and one of insanity.

In thirty-one subjects between the ages of 56 and 60 years, twenty-one, or 67.7 per cent., showed pial edema; eight moderate, nine marked and one extreme.

In thirty-four cadavers between the ages of 61 and 65, 82.5 per cent., or twenty-eight, showed pial edema; nine moderate, eleven marked and three extreme cases; one case of alcoholism.

In fourteen subjects between the ages of 71 and 75 years, 92.88 per cent., or thirteen, showed pial edema; six moderately and four markedly.

The four subjects between the ages of 76 and 80 years, 75 per cent., or three, showed pial edema, all markedly. The brain of the subject without edema was normal in size and appearance.

In six subjects between 81 and 85 years of age, 100 per cent. showed pial edema; two moderately, two markedly, and two extremely.

There was one subject showing pial edema between the ages of 85 and 90 years.

Nutrition.

In neither adults or children did there appear to be any definite relation between pial edema and emaciation.

Of 357 cases of pial edema in adults, forty-nine were not fully described as regards nutrition and therefore were subtracted from the general total, leaving 308 cases for analytical purposes. The state of nutrition in these cases of pial edema was as follows:

Fat	14, or 4.5 per cent.
Fair to good nutrition	168, or 54 per cent.
Poorly nourished	34, or 11 per cent.
Emaciated	64, or 20.7 per cent.
Markedly emaciated	28, or 9 per cent.

From the above it is seen that pial edema is associated with good nutrition in the majority of instances (50 per cent., as against 41 per cent. for the badly nourished).

Of the eighteen infants who had pial edema, two were not fully described as regards nutrition. The remaining sixteen presented findings similar to those of the adult series given above. The nutrition in these cases was as follows:

Fat	1
Well nourished	6
Poorly nourished	1
Emaciated	6
Much emaciated	2

The question whether all poorly nourished people have pial edema is answered by referring to the data concerning the cases in my series with dry

pias. In this group cachexia was present ninety-eight times unassociated with any subarachnoidal collections of fluid—a number sufficiently large to show that no relationship exists between the two conditions.

Relationship Between Pial and Edema Disease Processes (68).

The records were grouped according to the anatomical diagnosis with a view of determining whether any diseases or conditions were regularly associated with pial edema.

Pial edema is found on the average in practically two out of every three adults at autopsy. In our series of 542 adults, the subjects with pial edema, 355 in number, constituted 65.5 per cent. of the total number. Those without pial edema, 187 in number, comprised 34.5 per cent. of all the subjects.

It is evident that pial edema cannot be assumed to be related to any particular disease process unless it can be shown that it occurs with greater frequency with that disease than it does with other diseases. The simplest method to determine whether such an association exists for any particular disease is to compare the number of subjects which show pial edema with those which do not show pial edema. If the ratio exceed 65.5:34.5 (the ratio of our total cases as shown above), such a relationship would be indicated.

The total number of cases of a given disease from both the edema and the non-edema column was set down and the percentages computed. For example, under the heading "Adhesive Pleurisy" I found thirty-one cases associated with pial edema and twenty cases unassociated; the proportion of one to the other expressed in percentages is 64.5 and 35.5 per cent. respectively.

These figures represent the absolute percentages; since the number of cases of pial edema available for study is much larger than the number of cases without pial edema we can determine the relationship which the disease bears to the disease process in question by the use of a method which does away with the necessity of directly contrasting two unequal series. This is accomplished by reckoning percentages in each column (i. e., the pial edema, and the non-edema column) separately and then by comparing the percentages so obtained. The results in each case are found to be independent of the number of cases in either series, as they represent proportions only. Their value consists wholly in their use for purposes of comparison.

Using this method we reckon first the percentage of adhesive pleurisy (thirty-one cases) in the pial edema column (355 cases); this gives us the figure 8.7 per cent. We then similarly reckon the percentage of adhesive pleurisy (twenty cases) to the 187 cases of the "control" series which is found to be 10 per cent. These percentages being compared with one another indicate that adhesive pleurisy occurs with about the same frequency both with and without pial edema, since the proportion of occurrence of the lesion is nearly the same in either series. If now we should find that for any given disease we had a high percentage in the pial edema column, while much lower in the non-edema column, we should infer that the lesion either played some etiological role or was at lease definitely associated with pial edema. If on the other hand we should find the percentage in the pial edema column level with or below the percentage for the same disease in the other column we should decide that the disease bore no particular relation to pial edema.

It will be at once observed that a negative result is attained when the percentages for a given disease are the same or nearly the same in the pial edema and the control columns.

It was considered best to draw conclusions only where the figures were high enough to lend value to such percentages and for this reason lesions that appeared a few times only on the chart were not emphasized.

As a result of my preliminary studies in children and young people, it seemed wise to omit all cases under maturity from the disease columns, since it is possible that in them certain unexplained factors of development result in confusion.

Acute Parenchymatous Nephritis.—Reckoning the percentages by the method already explained, I found that acute parenchymatous nephritis was present in 10.9 per cent. of all cases with pial edema, and in 10.6 per cent. of all the cases of the series without pial edema.

Chronic Interstitial Nephritis.—Reckoning the percentaages in each column separately, chronic interstitial nephritis was found present in 32.9 per cent. of all cases with pial edema and in 30.4 per cent. of all cases without pial edema. The small granular kidney was present in 18.5 per cent. of all subjects having pial edema and 15.5 per cent. of all subjects without pial edema.

These figures do not indicate any essential relationship between pial edema and this type of kidney lesion (69).

Taken altogether our figures indicated an absence of association between pial oedema and acute or chronic Bright's disease.

Syphilis.—Aside from paresis, which will be referred to again, the figures of the table did not suggest that general syphilitic infection plays a causative role.

Chronic Pulmonary Tuberculosis.—This was present in 16.3 of all cases of pial edema and in 26.7 per cent. of all cases without pial edema.

Chronic Cardiovalvular Disease.—The figures obtained were 10.9 per cent. with pial edema and 11.7 per cent. without.

General Arteriosclerosis.—The figures obtained were 12.9 per cent. with pial edema and 9.4 per cent. without pial edema. These figures do not suggest a relationship between pial edema and arteriosclerosis.

Anemia.—We had only four cases of anemia. Two of these were associated with pial edema and two were not.

Advanced Alcoholism.—Here was found the first really significant evidence of relationship between pial edema and a disease process. Altogether there were eighty-nine cases of chronic alcoholism in the series. Of these seventy-two subjects had pial edema, or 80.9 per cent., while the remaining seventeen had dry pias, or 19.4 per cent. Reckoning the percentages for each column separately I found that chronic alcoholism was present in 20.2 per cent. of all our cases with pial edema and present in only 9 per cent. of all cases without pial edema. The frequency with which pial edema occurs in chronic alcoholism is thus represented by the ratio 20.2:9, the latter figure being taken to represent the proportion of chronic alcoholics in which no pial edema is present. It may thus be said that pial edema occurs in something like two-thirds of all cases of chronic alcoholism.

Pial Edema Associated with Transudation in Other Parts of the Body.—A. Localized or Generalized Subcutaneous Edema: My tables showed:

1. Localized edema of the subcutaneous tissues occurs apparently without special relation to pial edema.

2. Generalized edema: Anasarca showed a slight preponderance in the pial edema column, but the small number of cases quoted rendered this proportion of little or no value.

3. Ascites: The same thing may be said of ascites as of anasarca.

B. Polyserositis: I had a sufficient number of cases (forty-four) to warrant me in drawing conclusions from the percentages obtained. The percentage of

serositis cases was slightly higher in the control column (20 per cent.) than in the pial edema (18 per cent.). We therefore concluded that polyserositis is not associated with pial edema regularly and that it does not have necessarily the same causative factors.

Condition Associated with Acute Toxemia.—I had 466 cases which were considered as belonging in this category; 304 of these presented pial edema; the remaining 162 were not associated with this condition.

Reckoning the percentages of each series as already explained, I found acute toxemia in some form present in 85 per cent. of all cases that had no pial edema.

Conditions Associated with Bacteriemia.—There were 191 cases that could properly be grouped under the heading of bacteriemia; 111 of these presented pial edema, the remaining eighty had no pial effusion. I thus found bacteriemia present in 31 per cent. of all our cases of pial edema, and present in 42 per cent. of cases without pial edema.

Chronic Inflammatory Lesions in the Serous Cavities.—I had altogether 104 cases that were included under this heading (70); sixty-four of these showed pial edema, while forty were unassociated with pial edema.

Chronic inflammation of the serous cavities was thus present in 18 per cent. of the cases of pial edema and present in 21.3 per cent. of the cases without pial edema.

Chronic Gastritis.—This condition was present in 13.2 per cent. of the cases with edema of the pia and in 13.9 per cent. of the cases which did not present pial edema

Malignant Tumors.—The figures for these cases disclose nothing of significance.

Status Lymphaticus.—Of the fifteen cases of status lymphaticus in our series, twelve brains were large enough to completely fill the skull cavity. Pial edema was present in only three.

From the considerable proportion of large or fully developed brains noted with status lymphaticus, it might be inferred that we had here to do with a brain hypertrophy similar to that indicated by Anton (71) and described by Bartel (72) in some of his "status" cases. Such an inference should not receive undue emphasis; first, because complete weight records are not available in our series, and, second, because a large proportion of our subjects happened to be young people in whom brain volume is usually considerable.

Insanity.—The psychopathic cases, most important because of the possibility or organic changes being present, were too few in number for comparative figures.

Summary.

Age.—Edema of the pia was not found in my series before the fourth month. It occurred with moderate frequency from the fourth month to the fifth year. Between the fifth and sixteenth years it was absent. Beginning with the sixteenth year it rapidly increased in frequency to the period between the thirty-sixth and fortieth years (73), when, following a slight sharp decline, it continued to increase though less rapidly throughout the later periods of life, becoming universal in old age.

Nutrition.—Pial edema occurs independently of changes in the nutrition of the body.

Disease Conditions.—1. Pial edema does not appear to bear any particular relationship to the following common conditions: chronic nephritis; chronic pulmonary tuberculosis; chronic cardiovalvular disease; acute toxemia; bacteriemia; chronic inflammatory lesions in the serous cavities, and chronic gastritis.

2. Pial edema is definitely associated with chronic alcoholism, occurring in more than two thirds of all cases.

3. There were too few cases of syphilis, anemia and insanity to permit me to form conclusions in regard to these cases.

In conclusion I wish to emphasize, first, the progressive increase of pial edema after puberty; second, the absence of any relationship between pial edema and nutritional changes in the body at large; third, the negative findings as to association between pial edema and the general disease processes studied, except alcoholism.

Part 2. The Mechanics and Chemistry of the Spinal Fluid and the Anatomy and Physiology of the Membranes.

Having determined the relationship which pial edema bears to most of the important diseases, we are now in a position to study the character of the fluid present in this condition.

Normal Distribution of Cerebrospinal Fluid.

The cerebrospinal fluid is present within the confines of the subarchnoid space, the ventricles of the brain, the perivascular spaces and the central canal of the spinal cord. It is limited externally by the arachnoid membrane (external to which, over the cortex, lies the subdural space) and internally by the pia.

Distribution of the Fluid in Pial Edema.

In pial edema we find that the distribution of the fluid is the same as above stated, but that it is greatly increased in amount. The peculiarities as regards localization are as follows:

1. It is constantly found on the superior surface of the cerebral hemispheres, especially over the vertex.

2. It is found on the superior surfaces of the frontal lobes in relatively less amount.

3. The anterior and posterior cisternae are distended.

4. It is found often as bleb-like accumulations on the posterior and inner margins and on the superior surfaces of the cerebellar hemispheres.

5. There is occasionally a similar accumulation on the anterior poles of the temporosphenoidal lobes.

6. There is occasionally an apparent increase of fluid in the perivascular spaces.

7. There is occasionally considerable moisture of the brain tissue.

8. There is occasionally increase of fluid in the subarachnoid space of the cord.

9. Usually there is an increase of fluid in the lateral ventricles.

10. It is rare to see any increase over the lateral regions of the cerebral cortex.

No increase of fluid has ever been observed by me in the following places:

A. The under surface of the frontal lobes.

B. The under surface of the temporosphenoidal lobes.

C. The inner surface of the cerebral hemispheres.

We thus observe a general tendency on the part of the excess fluid to occupy a position above that of the brain tissue proper.

The Apparent Significance of This Distribution.

The localization of the fluid as given above is in marked contrast to that of the exudate in most cases of suppurative meningitis, especially in the epidemic type. As is well known, in most cases of meningitis the exudate is found chiefly at the base—a phenomenon that is especially noticeable in tuberculous and epidemic meningitis. The experience of this laboratory is fully in accord with the above statement and points to a tendency on the part of inflammatory changes in the meninges either to begin at the base and spread thence over the cortex, or to remain localized at the base (74).

The above localization of the fluid of pial edema is in itself suggestive of the action of gravity—and it is the one which the fluid would take were there any additional room within the cranial cavity, for the reason that as the specific gravity of the brain (1,038) is greater than that of the fluid in which it lies (1.002:1.010), the brain then would naturally seek a dependent position, forcing the fluid to a higher level.

The presence of the fluid in the cisternae pontis (ant. subarachnoidean space) has already been mentioned.

In performing an autopsy, however, some care is necessary in order to see the fluid in situ; for although the arachnoid at these points is normally somewhat thicker, it is at the same time unsupported and thus is readily torn when the brain is lifted up, allowing its contents to drain away into the cranium.

When the anterior subarachnoid space is ruptured, the fluid from the posterior immediately drains across the crurae cerebelli and thence into the skull cavity.

To what degree evacuation of the lateral ventricles follows this draining through rupture of the cisternæ is difficult to say, but that it is relatively slow there is every reason to believe.

The evacuation of the fluid over the cortex after removal of the brain also takes place gradually, apparently because the septa of the subarachnoidean chambers offer a partial obstruction to the flow of the fluid. In some cases the arachnoid reveals an almost g latinous mass but possibly this may be due to clotting of the fluid after removal.

That the fluid over the cortex is enmeshed is shown by the following facts:

1. The fluid over the cortex may remain in situ some time after the brain is removed.

2. Puncturing the dependent areas over the cortex sometimes results in the evacuation of only a small area about the site of the puncture. To illustrate this fact we may cite the following:

Case 1879.—Pia arachnoid moderately distended with clear slightly yellowish fluid, most marked over the convexity. A small puncture (about 5 mm. in diameter) was made over one of the sulci, the probe passed down through some watery and slightly gelatinous tissue to the cortex. The fluid about this puncture drained away leaving a depressed area 1 cm. in diameter, but the fluid beyond this area was apparently unaffected at the end of an hour.

The best way to maintain the cisternae intact, according to my experience, has been to insert the calvarium hook beneath the occipital protuberance and tear off the calvarium from below upward, instead of from above downward (the usual practice). The dura being then cut away, the frontal lobes can be lifted up disclosing the cisternae unruptured.

The pathologist has usually considered that an excessive amount of fluid at the base and the distention of the cisternae was an indication of an increased or high intracranial tension during life. Although it may be granted that this

is the explanation for a large number of cases, we must not forget that it is not conclusive evidence. The fallacy of conclusions based on such evidence is well illustrated by the conclusion drawn by the ancient Greeks on post-mortem evidence alone that the aorta contained blood mixed with air.

Thickenings of the Arachnoid.

In a considerable number of all pial edema cases there is a greater or less degree of opacity and thickening of the meninges. Sometimes this is chiefly limited to the area over the superior surface of the cortex along the longitudinal sinus; again it is found over cortex and cerebellum, and occasionally it has an even more generalized distribution.

In every case examined by me this opacity was found to be due to connective tissue thickening of the arachnoid, involving slightly the pia-arachnoid trabeculae, the pia itself showing no change.

This lesion consists of a proliferation of the fixed connective tissue elements without exudation or vascular changes.

That a process of this essentially proliferative character arising at a distance from the blood-vessels (75) should be the cause of the large fluid accumulations seems hardly possible.

Chemistry of Fluid in Pial Edema.

The final determination as to the inflammatory or non-inflammatory character of the fluid rests in the last analysis with the chemical examination. It is a well-known fact that fluid collected after death is worthless for examination, since post-mortem changes render chemical tests of doubtful value (76). The most satisfactory method is to collect the fluids intra vitam and to verify the findings thus obtained at autopsies. Such procedure, naturally, is attended with many disappointments.

With the exception of one case reported by Dufour (77), I have so far been unable to find in the literature any satisfactory record of analyses of spinal fluid from subjects of "wet-brain." A specific test for serum albumin or globulin rather than for protein should be of value in determining the question of possible inflammatory influences. Serum albumin is not a normal constituent of cerebrospinal fluid (78) and therefore its presence in the class of cases under discussion should indicate an exudative or transudative process. The absence of traces of serum albumin, in the light of our present knowledge of the secretory origin of the cerebrospinal fluid, would therefore indicate that the spinal fluid is the product of normal secretion. Professor Hastings has kindly supplied me with a series of analyses on this class of cases, and these will now be considered (79).

A spinal fluid from a subject of alcoholic "wet-brain" examined by him showed a protein content of 0.005 per cent., a minimum normal figure.

The figure for protein quoted above speaks strongly against the presence of either transudate or exudate in the spinal fluid of these cases. Professor Hastings analyzed several other specimens of alcoholic "wet-brain" without reference to the protein content.

The amounts in these cases ranged from 25 to 88 c.c.; all specimens were clear, sterile and free from coagulum or sediment. The cytological count was as given in Table 1.

Table 1.—Cell-Count—100 Fields.

Case	Polys.	Small Monos.	Endothelial Cells	N. B. Cs.
1	0	2	0	12
2	0	7	1	18
3	0	6	0	27
4	0	1	2	1,456 *
5	0	1	0	0
6	0	3	0	0
7	0	1	0	1
8	0	0	0	0

The conclusions from these analyses were that the fluid of alcoholic "wet-brain" did not differ from normal cerebrospinal fluid.

Circulatory Changes in the Membranes With Pial Edema.

It is well known that it is difficult to determine from post-mortem findings the circulatory conditions which were present during life in cases of pial edema, since alterations in the vascular system of the brain occur frequently just prior to and at the time of death.

My findings in regard to the condition of the meningeal circulation in pial edema (from post-mortem study of 100 cases) are as follows:

	No. of Cases
1. Active congestion of membranes, brain normal	12
2. Passive congestion of membranes, brain normal	21
3. Active and passive congestion of membranes, occurring together, brain normal	12
4 Active congestion of membranes with anemia of brain	1
5. Passive congestion of membranes, with anemia of brain	1
6. Active congestion of membranes and brain	5
7. Passive congestion of membranes and brain	7
8. Active and passive congestion of membranes and brain	10
9. Membranes and brain normal	20
10. Pia-arachnoid normal, brain anemic	4
11. Pia-arachnoid normal, brain congested	1
12. Anemia of brain and membranes	6

The commonest condition found was thus passive congestion of the membranes (present in 21 per cent.). The next most common was an apparently normal condition of the vessels of the brain and meninges (20 per cent.). Active congestion of the membranes and a condition of active and passive congestion of the membranes, occurred in 12 per cent. Anemia of the membranes and brain was present in 6 per cent. Altogether passive congestion of the membranes (either along or combined) occurred in fifty-one cases (51 per cent.), whereas active congestion was present in only forty cases (40 per cent.). The preponderance of passive congestion in this series agrees with the statement of Kaufmann (80) that most of the cerebral congestions observed at autopsy are of the passive type.

The frequent occurrence of passive congestion can hardly be taken as an indication that passive congestion has any special influence in the production of pial edema because there is nothing to show that it is more common with

* First-tube contaminated with blood.

pial edema than with the general run of autopsy cases. Furthermore, the wide variety of other vascular conditions cited in this series shows that pial edema bears no constant relation to any particular circulatory condition as seen at autopsy.

Part 3. The Physics and Mechanics of Pial Edema.

My observations thus far have been with a view to determine under what conditions pial edema is found. Let us now consider from a more theoretical standpoint the mechanics involved in the causation of accumulations of fluid in the pia-arachnoid. Two hypotheses present themselves:

1. The accumulation of fluid is the result of an increase of secretory activity of the chorioid plexus, or a lack of resorption, leading to a compression of the brain substance, or

2. The fluid has simply collected as a result of some undetermined process leading to a reduction in size of the brain.

The first hypothesis to be discussed is whether the fluid can actually compress the brain so as to give rise to the appearances met with in pial edema.

Before doing so, however, we must review certain well-established mechanical principles, a correct understanding of which is essential to a comprehension of some of the more complex problems in connection with pial or cerebral edema and serous meningitis.

It will not be necessary to sketch the outlines of the study of intracranial mechanics since the time of the second Monro (81). Let me present only the points that are involved and endeavor briefly to show their bearing on our subject.

Of fundamental importance is the old question "Is the craniospinal canal independent of the early influence of atmospheric pressure?"

Certain early writers, as Munro, Kellie (82), Abercrombie and Reid (83), and nearly all modern writers, among whom may be included Naunyn and Schreiber (84), Falkenheim and Naunyn (85), Horsley and Spencer (86), Horsley (87), Spencer (88), Roy and Sherrington (89), Hill (90), Kocher (91), Cushing (92) and many others, have decided that it is.

The earlier negative conclusions of Burrows (93) and Donders (94) were seriously considered for over half a century after their promulgation (95).

Leonard Hill's argument in favor of the absence of atmospheric pressure within the skull cavity was as follows:

If the spinal cord of a dog be divided or the splanchnic nerves be cut and the animal be placed in the vertical feet-down position the blood-pressure in the brain will under the influence of gravity fall to zero. If the skull be now trephined and the dura be rapidly opened the brain which was before in close apposition with the dura, may now be seen collapsing under one's very eyes, as it is emptied of blood by atmospheric pressure (96).

Additional evidence in confirmation of Leonard Hill's view that atmospheric pressure was absent in the skull cavity was obtained by the following experiments:

Experiment 1.—A trocar was introduced into the spinal canal of a prone intact cadaver and a small amount of fluid ran out owing to persistence of the pressure existing during life. No more escaping, suction was applied by means of a syringe and a little more fluid was obtained (altogether about 3 c.c.). The trocar was then removed and the calvarium opened. The cisternae contained the usual amount of fluid and likewise the spinal canal.

Experiment 2.—Calvarium removed first. Cisternae intact and seen to be full of clear fluid. Spinal puncture; 12 c.c. fluid came through cannula as if

under slight pressure. Cisternae re-examined and found full as before. Brain covered with wet cloths; in ten minutes fluid at the base had all disappeared into the cord.

The first experiment shows that outside air-pressure does not influence the contents of the craniospinal canal, since it was impossible to evacuate the fluid (except that in excess, and a slight amount in addition, due, possibly, to the stretching inward of the spinal membranes) through the single opening.

When, however, as in Experiment 2, a second opening was made and outside air-pressure admitted, there was no obstacle to the ready evacuation of the fluid in the direction of gravity.

The principle involved in these experiments is very easy to demonstrate with suitable apparatus. It will be observed that the fluid in the craniospinal canal in the first case has behaved as might be expected were atmospheric pressure absent.

There are certain anatomical features that require consideration in this connection.

Figure 2 represents the craniospinal canal, to all intents and purposes a closed cavity; the heavy black line its bony confines; the thinner line within represents the arachnoid which determines the outer limit of spinal fluid. While this membrane is not actually adherent to the overlying dura and bony walls, it lies in close apposition within the skull and is therefore incapable of any distention outward. In the spinal canal, however, it (together with the dura) lies like a sac at some distance from the bony walls, being separated from these walls by loose and compressible areolar tissue containing a plexus of veins (97).

Any increase of fluid within such a cavity must find room through distention of the sac outward (98).

It will be readily seen that while fluid can, at the outset, be forced into this sac without greatly raising intracranial pressure, after an amount has been forced in sufficient to crowd the tissue of the surrounding space and stretch the membranes considerably, there will be an ever-increasing resistance for each cubic centimeter forced in, until finally a point is reached at which no more fluid can be introduced, owing to the surrounding tissues having reached their limit of compressibility. It is the elastic recoil of these membranes which are put on the stretch, and the adjacent tissues which are compressed that, to a certain extent, gives the measure of intracranial tension, and thus when a cannula is introduced into this distended sac the tension of the tissues and membranes causes the expulsion of excess of all fluid, until the pressure is relieved.

Not all of the fluid, however, drains away, for a certain amount is necessary to fill various areas in and about the brain substances and the spinal cord. In

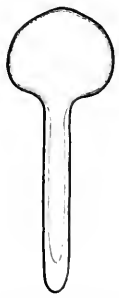

Fig. 2.—Diagram of craniospinal canal; heavy black line, bony confines; thinner line within, the arachnoid which determines the outer limit of spinal fluid.

the skull cavity the amount of this fluid scarcely varies since it cannot increase against the bony walls, nor can it diminish in amount anywhere without producing a vacuum at that point.

In the canal, on the other hand, the sac will retain only sufficient fluid to maintain equilibrium; that is to say, although a slight excess positive pressure is normally maintained, enough fluid will run out of a cannula to reduce this slight positive pressure to zero. Zero does not mean absence of fluid, but merely that the pressure within the spinal canal has dropped to the normal air-pressure.

Having considered the relation of atmospheric pressure to the skull cavity, and obtained some idea as to the relationship which exists between the membranes and the phenomena of intracranial tension, we may now go a step further and attempt to determine the effect of the factors just discussed on the brain itself. The difficulties involved in a study of this phase of our problem are all the greater when we realize that we know very little about two essential factors, i. e., first, the density or compressibility of brain tissue, and second, the permeability of the brain to fluids under pressure.

Our task now is to determine whether, according to the laws of physics and hydraulics, it would be possible for the brain (its blood-vessels excepted) to suffer compression through general increase of intracranial pressure.

The subject of compressibility must first be considered. Compression, broadly speaking, is brought about through expression of content. It is true that molecules are very slightly compressible, but, as indicated below (99), reduction in size from molecular compression would be so insignificant as to deserve but little attention.

For example, take a dry sponge; it is readily compressed in the hand through expulsion of the air molecules from its chambers. On being allowed to soak up water it gains in size and weight but still remains compressible through the ready loss of the water under pressure. If the same saturated sponge be now placed in an air-tight rubber bag which fits snugly at all points, compression is no longer possible, since there is now no longer any means of escape for its fluid contents.

The same principle applies to all homogeneous semifluid bodies; compression takes place by expression of air or fluid content where possible but when air or fluid cannot be expressed the body is termed incompressible.

Since, then, compression sufficient to be visible can only take place through expression of content, it is only fair to inquire whether such compression could occur within the skull cavity. An attempt to force fluid into a receptacle already filled with a solid or fluid would not result in an increase in the amount within the receptacle, but its molecules would simply transmit the added pressure to the walls and there is no reason to assume that the brain is to be looked on as an exception to this physical principle. Compression cannot take place without corresponding allowance being made for the displaced substance in the enclosing medium. Lacking this allowance any reduction of size is impossible.

A converse process, i. e., brain swelling, requires a corresponding displacement of content that is under ordinary circumstances allowed for by the evacuation of spinal fluid—a process that takes place when tension is somewhat raised.

While there is, according to the above explanation, no ground for the belief that general high intracranial pressure could bring about visible changes in the size or contour of the brain, it is barely possible that pressure may be a factor in inducing molecular compression, for although such molecular compression as might result from those degrees of intracranial pressure that have been registered clinically are very slight when judged from the standpoint of the physicist,

still so sensitive a tissue as the nerve cell is known to be, might appreciate the very small increases of molecular densities brought about in this manner.

We are not, however, justified in speculating on the chances of molecular compression within the brain until more is known about the compressibility of gray matter; if, for instance, this is slight, pressure would be transmitted through the brain to the surrounding bony confines, the intervening cells of the gray matter being but little affected. On the other hand, were the compressibility greater, pressure would not be so readily transmitted and a slight amount of molecular compression would doubtless result.

The influence of pressure from general causes has already been pointed out; we may now briefly contrast with these the effects of pressure due to local conditions, namely, those caused by hemorrhage or due to tumor growth.

The multiplication of tumor cells exerts, as in other organs, its first effect locally—namely, in compression and pressure-atrophy of the adjacent tissues and in compression of the adjacent vascular structures in the brain. Further compensation for any increase in growth or hemorrhage develops at the expense of the spinal fluid, which is resorbed, while when intracranial tension exceeds that in the venous structures the latter become somewhat compressed (100).

Striking post-mortem evidence of compression through any intracranial growth is seen in the marked flattening of one or both hemispheres, the dry pia and the marked diminution in amount of blood found in the pial vessels.

For these reasons I believe that compression of the brain by the fluid in cases showing pial edema is an impossibility, and that the appearances observed in cases of pial edema are not attributable to the pressure of the fluid. These appearances are thus the result of some other agency, in all probability they represent a redistribution or replacement process.

Part 4. Physics and Mechanics of Pial Edema.

For the purpose of determining whether or not I had to deal with a replacement process, I began the rather elaborate system of post-mortem observations which are now to be described.

The cases on which these observations were made were taken from the general autopsy service of Bellevue Hospital. The technic was as follows:

Technic.

When possible the calvarium was removed before the thorax was opened in order to prevent a redistribution of the blood. Many times, however, it was impracticable to halt the general autopsy work for this rather considerable interval and the result was that when the thorax was opened and the large veins were cut, as soon as the calvarium was opened and the relative negative pressure within the skull cavity removed, a considerable amount of blood would drain into the thorax from the vessels of the head.

The chief effect of this loss of blood was a reduction in the volume of the brain, causing a discrepancy between the volume of the cranial cavity as afterward registered and the total measured cranial content. The influence of this factor on the weight, specific gravity and volume of the brain, however, need not be considered, as when the blood was not evacuated into the thorax, it would be lost as the brain was being removed, and thus would not enter into subsequent computations.

As soon as the calvarium was removed, a pan was placed beneath to catch the drippings, and the tentorium carefully cut away and laid aside. The hemispheres were gently pushed apart along the great longitudinal furrow, and a

TABLE 2.—CASES ARRANGED IN GROUPS ACCORDING TO AMOUNT OF EDEMA PRESENT.

Group 1.—Cases Having No Edema.

Acc. No.	Age.	Anatomical Diagnosis.	Amount of Fluid in Ventricles (c.c.).	Specific Gravity of Cerebrospinal Fluid.	Weight of Brain (gm.).	Specific Gravity of Brain.	Volume of Brain (c.c.).	Volume (c.c.) of Excised Dura (Tentorium and Falx).	Total Fluid in Cranium (c.c.).	Total Capacity of Cranium (c.c.).	Discrepancy Between Volume of Brain with Membrane and Capacity of Skull (c.c.).
1,974	35 yrs.	Polyserositis	2	Contam.	1278	1.035	1234	30	26 c.c.	1290	26
2,099	5 mos.	Fatty liver; encephalon siccum.....	0	Contam.	576	1.033	558	0	30 m.	565	7
2,105	44 yrs.	Status lymphaticus	3	1.108	1322	1.039	1272	28	20 c.c.	1345	53
2,146	46 yrs.	Carcinoma of stomach	2	Contam.	1000	1.046	954	18	30 c.c.	1000	28

Average age (infants excluded), 39.5 years. Average specific gravity of brains, 1.0363.
Average discrepancy, 28.8 c.c. This figure represents about the normal quantity of spinal fluid found in the skull with the brain at its maximum stage of development.

Group 2.—Cases with Slight Edema.

1,959	43	Delirium tremens; chronic alcoholism.....	2	Contam.	1275	1.044	1218	20	30	1280	32
2,107	21	Chronic pulmonary tuberculosis.....	3	Contam.	1410	1.036	1362	30	50	1445	53
2,130	42	Chronic pulmonary tuberculosis.....	27 Clear	1.008	1275	1.039	1227	20	57	1330	83

Average age, 35.3 years. Average specific gravity of brains, 1.0397. Average discrepancy, 53 c.c.

Group 3.—Cases with Moderate Edema.

Acc. No.	Age (Years).	Anatomical Diagnosis.	Amount of Fluid in Ventricles (c.c.).	Specific Gravity of Cerebrospinal Fluid.	Weight of Brain (gm.).	Specific Gravity of Brain.	Volume of Brain (c.c.).	Volume (c.c.) of Excised Dura (Tentorium and Falx).	Total Fluid in Cranium (c.c.).	Total Capacity of Cranium (c.c.).	Discrepancy Between Volume of Brain with Membrane and Capacity of Skull (c.c.).
1,963	50	Cardionephritic	6	1.007	1401	1.030	1359	29	65	1560	72
2,057	37	Brain tumor	Clear 8	Contam.	1230	1.038	1184	22	53	1270	64
2,082	33	General miliary tuberculosis	Clear 12	1.007	1271	1,035	1227	50	106	1385	108
2,101	?	Chronic pulmonary tuberculosis	Clear 5	1.003	1044	1.040	1003	21	76	1100	76
2,106	21	Chronic tuberculous peritonitis	Clear 5	1.006	1115	1.038	1074	20	58	1225	131
2,113	40	Chronic alcoholism; gangrenous cervicitis	Clear 8	1.005	1268	1.038	1221	32	90	1380	127

Average age, 36.2 years. Average specific gravity of brains, 1.0365. Average discrepancy, 96.3 c.c.

Group 4.—Cases with Marked Edema.

1,964	54	Carcinoma of esophagus; bronchopneumonia	4	1.009	1347	1.037	1299	35	67	1510	176
2,039	56	Acute general miliary tb.; tb. of meninges	18	Contam.	1447	1.033	1400	29	100	1635	206
2,085	1	Marasmus; congenital diplegia	.	.....	668	1.030	646	10	Collected 100; remainder lost.	830	162
2,108	40	Chronic pulmonary tuberculosis	3	1.007	1194	1.038	1149	22	115	1340	169
2,131	67	General arteriosclerosis	24	1.007	1197	1.039	1151	30	125	1345	164
2,147	70	Chronic miliary pulmonary tuberculosis	Clear 18	.....	1335	1.038	1286	40	138	1550	224

Average age (children excluded), 57.6 years. Average specific gravity of brains, 1.0358. Average discrepancy, 183.5 c.c.

Group 5.—Cases with Extreme Edema.

2,052	66	Chronic interstitial nephritis	7	Contam.	1000	1.027	974	27	122	1190	189
2,148	61	Chronic tuberculous bronchopneumonia	Clear 25	.....	1231	1.041	1182	20	175	1430	228

Average age, 63.5 years. Average specific gravity of brains, 1.034. Average discrepancy, 208.5 c.c.

small incision made through the corpus callosum into each lateral ventricle, the contents of which were withdrawn by a pipette and measured.

The brain was then removed and allowed to drip into a pan until the watery fluid over the cortex and elsewhere had quite disappeared. It was then weighed in the air and its weight recorded. Following this it was weighed in water at 15 C. with the same balance and its specific gravity computed.

It is important to note here that considerable variation in specific gravity was unavoidable owing to the fact that in many of the brains it was impossible to get rid of all the spinal fluid which was retained in pockets and corners of the sulci. This increased the weight in air, but not in water, and as a very few grams weight will make quite a wide variation in specific gravity, it will be seen that I was here often confronted with an important source of error.

The brain proper having been disposed of, the drippings, the free fluid in the calvarium, and the ventricular fluid, were carefully collected and measured.

The portions of dura cut away were measured in water giving the number of cubic centimeters displaced, and, finally, the capacity of the skull cavity was measured by means of dried peas.

By the addition of the total fluid content of the calvarium with the dura and the brain volume (in c.c.), a fairly accurate estimate of the total cranial content is arrived at. Comparing this figure with that representing the cranial volume a very slight error is observed in those cases in which the cerebral blood was allowed to escape into the thorax previous to the examination of the head. In the latter cases the discrepancy represents this loss and can be readily identified by a study of the chart.

Having ascertained the above data, it was thought to be best for purpose of study to arrange the cases in groups corresponding to the amount of pial edema present.

On examining Table 2, in reference to the specific gravities, the recognized tendency for brains to become lighter at the extremes of age is observed.

There is, however, a most important point which these tables have clearly brought out; i. e., that there is a gradually increasing discrepancy between the capacity of the brain cavity and the volume of the brain, which is directly proportional to the degree of pial edema present.

In Group 1, cases without pial edema, we see that the average discrepancy is only about 29 c.c., this figure being presumably the equivalent of the cerebrospinal fluid present. In these cases the brain may be assumed to fill the skull cavity within physiological limits.

In Group 2, where the edema is slight, there is an average total discrepancy of 53 c.c., an increase of 24 c.c. over the preceding class.

As the edema increases, there is a corresponding increase in the discrepancy. In the remaining groups it is, with moderate edema, 96 c.c.; with marked edema, 183 c.c.; and lastly, in the two cases of excessive edema in the series the discrepancy culminates with 208 c.c. as a maximum, this in spite of the fact that one of the cases examined had a very small skull. This discrepancy can, of course, be interpreted in only one way, i. e., that the brain has become so reduced in size as not to fill completely the cranial vault.

Were compression the cause of this reduction in size there should be a corresponding increase in the specific gravity of these brains, proportional to the degree of edema. This, we see, is not present; indeed, if it is permissible to say anything at all in this connection, it is that the specific gravity has fallen slightly with the higher grades of edema. To say merely that the condition is a hydrops *ex vacuo* does not, by any means, dispose of the whole subject; let us therefore resume the consideration of the data before us.

Leaving out of account the two cases cited, in very young children, we observe that the more vigorous periods of life coincide with the milder degrees

of discrepancy between brain and skull cavity, while the more marked differences are exhibited in the more advanced periods of life. It has been universally recognized that shrinkage of the brain is an accompaniment of extreme old age, but the extent to which this phenomenon reaches back into the years of vigorous manhood has not been generally considered.

Table 3 shows the brain weight at different ages; for children only, by Parrot (101), Pfister (102), and Marchand (103); through childhood up to the twenty-fifth year by Vierordt (104); at all ages Boyd (105), Meynert (106) and Handmann (107); and from the twentieth to the ninety-fifth year, Bischoff (108).

Table 3.—Brain Weights of Children and Young Adults*

Vierordt			Parrot			Pfister			Marchand		
	Weight (gm.)			Weight (gm.)			Weight (gm.)			Weight (gm.)	
Age	Males	Females	Age	Males	Females	Age	Males	Females	Age	Males	Females
Newborn .	381	384	.....	.....	.....	.....	340*	330*	.....	371	361
1 month...	463	402	.....	364	331	2-4 wks..	431	396	.....	411	375
2-3 mos...	548	527	.....	490	412	2 mos...	461	415	.....	474	450
4-6 mos...	632	575	.....	575	552	3 mos...	519	504	.....	612	587
7-9 mos...	733	771	7-11 mos.	778	719	4-5 mos..	583	562	.....	.....	.....
10-11 mos. ...		693	.....	.....	.....	6-8 mos..	733	666	7-11 mos.	796	802
1 year....	944	872	.....	.....	.....	9-10 mos.	786	684	.....	.....	.....
2 years...1,025		960	1-2 years	964	913	11-12 mos.	851	727	.....	967	893
3 years...1,112		1,040	.....	.....	.....	.....	958	901	1-2 years	1,011	896
4 years...1,327		1,138	2-4 years	1,167	1,063	.....	1,099	1,044	2-3 years	1,080	1,099
5 years...1,282		1,220	.....	.....	.....	.....	1,183	1,091	3-4 years	1,310	1,024
6 years...1,353		1,258	4-6 years	1,261	1,137	.....	.....	.....	4-5 years	1,273	1,183
7 years...1,348		1,295	.....	.....	.....	.....	.....	.....	5-6 years	1,343	1,245
8 years...1,366		1,150	.....	.....	.....	5-8 years.	1,219	.....			(at 5 yrs.)
9 years...1,425		1,242	.....	.....	.....	.....	.....	.....	.....	.....	.....
10 years..1,417		1,267	.....	.....	.....	.....	.....	.....	6-9 years	1,360	1,242
11 years..1,379		1,238	.....	.....	.....	9-12 years	1,285	1,265	.....	.....	.....
12 years..1,415		1,245	.....	.....	.....	.....	1,289	.....	10-14 yrs.	1,346	1,221
13 years..1,475		1,255	.....	.....	.....	.....	.....	.....	.....	.....	.....
14 years..1,289		1,345	.....	.....	.....	.....	.....	.....	.....	.....	.....
15 years..1,471		1,235	.....	.....	.....	.....	.....	.....	.....	.....	.....
16 years..1,435		1,272	.....	.....	.....	.....	.....	.....	.....	.....	.....
17 years..1,409		1,236	.....	.....	.....	.....	.....	.....	15-19 yrs.	1,404	1,309
18 years..1,441		1,324	.....	.....	.....	.....	.....	.....	.....	.....	.....
19 years..1,384		1,234	.....	.....	.....	.....	.....	.....	.....	.....	.....
20 years..1,444		1,228	.....	.....	.....	.....	.....	.....	.....	.....	.....
21 years..1,425		1,319	.....	.....	.....	.....	.....	.....	.....	.....	.....
22 years..1,348		1,280	.....	.....	.....	.....	.....	.....	.....	.....	.....
23 years..1,402		1,277	.....	.....	.....	.....	.....	.....	.....	.....	.....
24 years..1,419		1,248	.....	.....	.....	.....	.....	.....	.....	.....	.....
25 years..1,428		1,230	.....	.....	.....	.....	.....	.....	.....	.....	.....

* Parrot's, Pfister's and Marchand's ages same as in Vierordt's column unless otherwise indicated.

Group 2.—Brain Weights at All Ages*

Handmann			Meynert			Boyd			Bischoff		
	Weight (gm.)			Weight (gm.)			Weight (gm.)			Weight (gm.)	
Age	Males	Females	Age	Males	Females	Age	Males	Females	Age	Males	Females
.....	239†	247¶	.....	.....	.....	Newborn.	393	347	.....	.....	.....
7 days...	404‡	377‖	.....	.....	.....	.....	.....	.....	.....	.....	.....
8-30 days	357	357	.....	.....	.....	1-3 mos..	493	495	.....	.....	.....
.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
2-3 mos..	485	486	.....	.....	.....	.....	603	560	.....	.....	.....
4-6 mos..	650	490	.....	.....	.....	7-11 mos.	777	709	.....	.....	.....
7-12 mos.	830	817	.....	.....	.....	1-2 years.	941	845	.....	.....	.....
2 years..	1,075	998	.....	.....	.....	.....	.....	.....	.....	.....	.....
3 years..	1,208	1,088	.....	.....	.....	2-4 years.	1,097	997	.....	.....	.....
4 years..	1,185	1,173	1-19 yrs.	1,114	.....	.....	.....	.....	.....	.....	.....
5 years..	1,245	1,225	.....	.....	.....	4-6 years.	1,140	1,137	.....	.....	.....
6 years..	1,215	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
7-9 years.	1,345	1,283	.....	.....	.....	6-14 yrs..	1,304	1,156	.....	.....	.....
10-14 yrs.	1,400	1,215	.....	.....	.....	.....	.....	.....	.....	.....	.....
15-17 yrs.	1,429	1,281	.....	.....	.....	15-19 yrs.	1,376	1,246	.....	.....	.....
18-19 yrs.	1,328	1,226	.....	.....	.....	.....	.....	.....	.....	1,396	1,234
20-29 yrs.	1,392	1,252	.....	1,306	1,169	20-30 yrs.	1,358	1,239	.....	1,365	1,233
30-39 yrs.	1,367	1,246	.....	1,326	1,167	30-40 yrs.	1,366	1,222	.....	1,366	1,240
40-49 yrs.	1,358	1,247	.....	1,317	1,173	40-50 yrs.	1,348	1,214	.....	.....	.....
.....	.....	.....	.....	.....	.....	At 50 yrs.	1,352	1,208	.....	.....	.....
50-59 yrs.	1,357	1,227	.....	1,240	1,174	50-60 yrs.	1,345	1,225	.....	1,375	1,200
60-69 yrs.	1,326	1,208	.....	1,289	1,161	60-70 yrs.	1,315	1,210	.....	1,323	1,178
70-79 yrs.	1,282	1,175	.....	1,206	1,098	71-80 yrs.	1,290	1,170	.....	1,279	1,121
80-84 yrs.	1,250	1,126	80-89 yrs.	.....	1,148	Over 80.	1,284	1,127	80-85 yrs.	1,023	942

* Meynert's age grouping follows Handmann's; and Bischoff's is similar to Boyd's except when otherwise indicated.

† Boys under 49 cm. length of body.
‡ Boys over 50 cm. length of body.
¶ Girls under 50 cm. length of body.
‖ Girls from 50 to 55 cm. length of body.

The highest figures reached in Vierordt's column, Table 3 (1,475 gm.) occur at the age of 13 years in males. Among the females the highest weight recorded is (1,345 gm.) at the age of 14. This table, however, can scarcely be considered complete enough for our purposes.

Boyd's figures give the period of maximum weight as between 15 and 19 years, with a subsequent loss up to the age of 80 of about 90 gm.

Bischoff represents an irregular decline from 20 to 30 onward in males. His highest figure quoted in females, however, is between 40 and 50.

Handmann finds that the greatest brain weight occurs in males between the seventh and fifteenth years and that after the twenty-ninth year there is a gradual and steady decline. In females, curiously enough, his highest recorded brain weight (1,283 gm.) occurs in rather young children, seventh to ninth years. There is a slightly lower figure given for the period between the fifteenth and seventeenth years. There is, however, no rapid decline evident until middle age.

Peacock gives the maximum weight as between 25 and 30, Broca (109) at between 30 and 35, while Meynert's figures indicate that loss of weight begins even later in life.

Blakeman (110) concluded that the human prime in brain weight seems to fall before the twentieth year and that after this period there is on the whole a continuous fall. Taken altogether these data afford clear evidence that recessive changes within the brain begin often quite early in life, although it is evident from the study of even such limited data as I present that in some cases the brain maintains its maximum volume until its possessor is well advanced in years.

From these data there may be noted a strong tendency on the part of the age curve of pial edema as plotted out from my series of 375 cases to coincide with a curve plotted out in accordance with a scale of loss of brain weight for equivalent ages, that is to say, as the curve for pial edema rises, the average of brain weights will be found to fall correspondingly from point to point on the chart.

Apart from the sudden rise in the pial edema curve between 30 and 40 years, due to the inclusion of the large number of alcoholics dying within this period, the evidence is strong that pial edema coincides with the reduction of brain weight that is common after puberty, and, such being the case, I infer that it is secondary to a reduction in size of the brain and that it therefore represents a replacement process due to the brain shrinkage.

Such a shrinkage is of course to be looked on as entirely physiological. The discrepancy between the skull and the brain is doubtless to a certain extent modified by the sensible shrinkage of the head diameters described by Blakeman (111) as occurring in general hospital cases, but this is really slight, being very roughly only about 5 mm. between the twentieth year and old age.

Pathological shrinkage of the brain, that due to contraction caused by disease, has long been recognized. Clapham (112) in 1873, Crichton-Browne, (113) 1879, and Mercier (114) in 1891 published observations on the subject of lessened brain weights in the insane drawn from large series of cases. Boyd's figures, with which Crichton-Browne agrees, are so tabulated as to be readily intelligible; they are given in Table 4.

Donaldson in considering this table (115) regards the last three on the list as those in which wasting of the brain takes place.

Table 4.—Brain Weight in the Insane as Given by Boyd*

Males			Females		
Diagnosis	No. of Cases	Encephalic Wt., gm.	Diagnosis	No. of Cases	Encephalic Wt., gm.
Mania	108	1,393	Mania	107	1,227
Recurrent mania	30	1,383	Recurrent mania	33	1,238
Melancholia	52	1,335	Melancholia	68	1,261
Epilepsy	89	1,310	Epilepsy	60	1,216
Dementia	49	1,307	Dementia	61	1,188
General paralysis	122	1,304	General paralysis	30	1,162
Senile dementia	29	1,259	Senile dementia	12	1,226

Kaufmann (116) describes general cerebral atrophy in connection with old age, prolonged illness, chronic lead poisoning, alcoholism and dementia paralytica. Our researches in connection with pial edema confirm the statements in regard to old age and alcoholism and indirectly disagree with that in regard to prolonged illness (117), but do not cover the other conditions mentioned.

Altogether I may summarize by saying that pial edema follows atrophy of the brain; first, in the great majority of instances as a result of physiological changes beginning at or soon after puberty; second, in a smaller group as the result of certain pathological conditions, notable among which are chronic alcoholism (118) and certain forms of insanity.

My earlier review and demonstration of the principles underlying intracranial pressure is justified, first, by the necessity of ruling out a hypothetical cause (namely, local compression) for the appearances noted in case of well-marked subarachnoidal edema; secondly, by the evident need of restating these principles to those observers on serous meningitis who have apparently overlooked them, and, lastly, by the necessity of reviewing the fundamentals before entering into such discussion of the more complex and minute phases of the question as is inevitable in a consideration of the relations that exist between the present work and the conceptions of the Traube school, the observations of Kolisko and the experimental work of Mott and of Cannon.

In connection with Mott's work, his findings of increased fluid in the skulls of general paralytics (119) are in accord with the well-recognized tendency of these cases to brain shrinkage and consequent hydrops ex vacuo, as already pointed out. It must not be forgotten that brain shrinkage usually implies a widening of the perivascular spaces similar to that seen in senile brains and graphically described by Gowers (120). Such a condition is frequently encountered at autopsy, and it is not strange that the excessive moisture found on section of the brain should often be erroneously ascribed to a dilatation of those spaces by serum transuded from the blood vessels. As a matter of fact it seems to be questionable whether dilatation of these "lymphatic" spaces is a possibility. Mott adheres to the view, based on microscopical observations, "that the whole brain is permeated by a canalicular lymph-system containing cerebrospinal fluid, the large processes of the neurons lying in lymph-spaces which are continuous with the perivascular lymphatics" (121).

If these observations are true, then dilatation of the perivascular canals by transudate from contained blood-vessels is impossible, since pressure exerted within them would be readily transmitted by the serum to the surrounding tissue and would therefore be everywhere equal and thus prevent such a phenomenon.

As a matter of fact there is some evidence to show that the perineuronal lymph-spaces are not so readily permeable as is generally believed. I have met striking lack of success in attempting to perfuse methylene-blue normal saline solution into fresh brains under varying degrees of pressure. Whether the apparent impermeability of the brain represents a true finding or whether this

* Crichton-Browne, Brain, 1879, i, 511; quoted from H. H. Donaldson's Growth of the Brain.

failure to demonstrate ready permeability was due to faulty technic or unconsidered factors of capillarity cannot be stated; the technic (122) was similar to that successfully employed by Brodie in demonstrating the permeability of other organs.

I shall not consider Mott's production of edema of the brain in animals by experimental ligation of arteries (121), since his description shows plainly that his conception of brain edema is widely divergent from that held by the Traube school.

His reference to the ease with which the perineuronal spaces were made out in some of the cases of experimental anemia (121) is of lessened significance when I remind the reader that these appearances are often due to artifacts. Also it must not be overlooked that Huguenin (123) has observed from experiments on animals that a given brain is moister after death than during life.

The essential point to be held in mind in connection with the transudate theory is that pressure continuity exists throughout all the fluid molecules in the cranium. Pressure caused by transudation from the blood-vessels would be transmitted from molecule to molecule everywhere throughout the cranial cavity and spinal canal—it would be felt, generally speaking, as fully or nearly as much in the subarachnoid fluid on the outside of the brain as in that within the perivascular and interstitial spaces. Under these circumstances there would be no disarrangement of the brain, that is to say, no crowding up of the external portions against the bony vault. Nor could there be, as already stated, any measurable molecular compression although intracranial pressure were raised to great heights. Vascular compression would occur, but this would not explain the soggy condition of the tissues (often considerable from the gross and the histological point of view) intervening between the vascular spaces.

On physical grounds therefore we are not justified in maintaining that transudation is a cause of cerebral edema, whatever role it may play in the production of edemas elsewhere (124).

The old idea propounded by Traube that cerebral edema is due to transudate has persisted with some tenacity in spite of the evidence to the contrary offered by Cannon (1901). With the exception of Cushing (1903 and 1908) (24, 25) it appears that the significance of Cannon's work has been largely overlooked.

Leyden (125), Duret (126), Cybulski (127) and Hill (128) all believed that intracranial tension must equal blood tension in order to produce death. Cannon (129), reasoning from their experiments, in an attempt to explain the mode of death after trauma of the brain, decides that transudation could not raise pressure sufficiently to bring this about, since pressure outside the vessels would not equal that within, owing to loss through resistance of the tissue.

The reader deduces for himself that since the accepted mode of death in cerebral edema is through high tension inducing arterial collapse, the cause of the cerebral edema cannot be transudation.

Cannon develops a theory of edema which differs radically from that of all other observers and in which he ascribes the swelling to osmosis induced by chemical changes in the brain-cells themselves. He finds a satisfactory analogy in the experimental work of Budgett (130) and of Loeb (131) on muscle tissue, and supports his arguments by observations on the capacity of brain tissue when deprived of its blood supply to take up water from a solution isotonic with the blood (132).

He lays no particular emphasis on causation but points out that Loeb and Budgett emphasized lack of oxygen as a probable cause of the chemical changes

in their experiment, and demonstrates the manner in which lack of oxygen would follow as a result of circulatory changes resulting from brain contusion.

Cannon's idea that lack of oxygen sets in motion the processes described, while satisfactory from surgical standpoint, does not seem to apply equally well to other pathological conditions.

His theory has the merit that it is the only one so far given that is unassailable from the standpoint of mechanics and I cannot but feel that the process described, or something like it, is responsible for all true cases of cerebral edema, meaning by this edema of the brain as distinguished from pial edema.

Cannon (133) suggests a seeming objection to the working out of his theory in the occurrance of fluid noted in the dura or in the ventricles in certain recorded cases; "for why," he says, "does not the fluid pass into the tissues rather than accumulate if the tissues have the great osmotic power attributed to them?" He believes that the question cannot be definitely settled until the osmotic pressure of the fluids has been determined; he adds that the fluids must be encapsulated and gives the following as a possible explanation:

The diffusion of salts from the injured tissues into even a slight amount of fluid in an encapsulated space would render that fluid of higher osmotic pressure than the blood-plasma. The plasma would thereupon pass into the encapsulated space in obedience to osmotic laws and thus increase the fluid in its compressing effect. Further change in the injured tissues would lead to greater swelling in them and to diffusion of more of the dissolved products of decomposition. The diffusion into the encapsulated fluid would still further increase its osmotic pressure, and result in still more plasma coming to increase its volume. Thus there would be a passage of salts from tissues to blood in a series of decreasing concentrations, and a passage of fluids to tissues in a series of increasing osmotic pressures. And since water will pass more rapidly than salts through membranes, the result is usually a greater and greater pressure until death supervenes.

The importance of emphasizing Cannon's frank objection to his own theory and his explanation of the same is that if unanswered it affects our own theories and conclusions as well. Such a subdural process as that to which he refers suggests in a way as an analogy, a subdural hydrocephalus—a process in regard to which we know extremely little. Fluid in any amount is so seldom seen post mortem (134) in the subdural space that we seldom expect to come across anything except the yellowish serous fluid that often accompanies a pachymeningitis hemorrhagica interna. Possibly this fluid may not be so readily absorbed as the normal spinal fluid of the subarachnoid space.

More frequently during life the subdural space seems to contain thin exudate of an apparent inflammatory origin, especially where there is middle ear disease. This exudate, or transudate, may or may not represent the incipient stage of a purulent pachymeningitis and has more than once been described by operating otologists as meningitis serosa. Considering these facts, it seems unwise to attempt to draw any conclusions as to why subdural fluid seems to remain in the subdural space in some cases of apparent brain edema when one might naturally expect that it would be squeezed out.

Cannon's explanation as to the action resulting from the diffusion of salts from injured tissues into an encapsulated space, while theoretically sound for dead membranes, ignores the fact that living animal membranes have a selective action permitting the passage of one ion or molecule and preventing the passage of others, and he also appears to overlook the fact that such diffusion would affect primarily the subarachnoid fluid which directly envelops the brain tissue.

We actually do not know the mode of absorption of the subarachnoid fluid nor what relation it bears to the subdural fluid, for experiments thus far conducted have not settled this point. Furthermore, were the resorption of the subarachnoid fluid interfered with, the type of swollen brain described by Traube and Kolisko could not occur since its change in volume must necessarily be largely at the expense of spinal fluid.

In regard to the occurrence of accumulations of fluid in the ventricles, of which Cannon speaks (133), several hypotheses may be offered; first, that the cases mentioned were not those of actually swollen or edematous brains; second, that if they were the edema was in its incipience; third, that there was some obstruction to the outflow of spinal fluid from the ventricles. It is conceivable that under certain conditions of acute brain swelling such a result might be brought about. The oft-described permeability of the brain does not always seem to hold good, for cases have occurred in our experience with apparent sudden onset in which flattened dry convolutions, dry cut section and distended ventricles afforded almost proof positive of a confined and increasing ventricular secretion.

A newer theory of edema than that offered by Cannon and one which applies equally well to intracranial conditions is that recently advanced by Fischer (134). This theory, built on experiments, assumes that certain alterations in the fluids of the body, as for example too great alkalinity or acidity (including excess of carbon dioxid) so increases the affinity of colloids for water that they become swollen with resultant edema and swelling of the tissues. Fischer cites as examples of this action of colloids, swellings due to stings of insects, the swelling of gangrenous tissues when water is applied to them, and the swelling of dead bodies kept in water, as cases in point. The brain is rich in colloidal substances, which, should they suddenly begin to absorb water (136), would increase its volume in such a manner as to force all the cerebrospinal fluid from the skull. A subsequent evacuation of a certain amount of fluid into the tissue spaces of the brain, according to the mechanism described by this writer, would give rise to the soggy appearance observed.

While the type of brain of which I have been speaking is undoubtedly the one which Traube's followers have had in mind in their discussions, it is evident that they have now and then (through oversight of a mechanical principle) confused the swollen edematous brain with the more common form in which there is extensive or moderate pial edema and more or less moisture of the brain substance—the type which has been demonstrated as a physiological or pathological brain shrinkage.

In the first place, although it is impossible for the brain and subarachnoidal fluid to be increased at the same time (127), certain of the cases are so described as to imply that this may have taken place. In Cushing's article (138) in connection with subtemporal decompression in a case of chronic nephritis which has already been referred to, we are told that there was considerable fluid in the subarachnoid space and that the arachnoid membrane was pricked to allow of this escaping. The obvious deduction on reading Cushing's article is that he was dealing with the type of brain which has been especially discussed in this paper, and not with a true case of edema of the brain, as he may have supposed.

Similarly Phelps (139) cites a case of a patient dying of traumatic brain injury (the age is not given) in which there was moderate subarachnoid effusion and well-marked edema of the brain. Cannon (140) cites this case as an example of brain edema. To multiply such instances is unnecessary.

The conclusion to which we are led, based on the fact that in over 1,000 autopsies taken from the records of the Bellevue Laboratory (141), there have

occurred no truly swollen or edematous brains of the types described either by Kolisko or by Traube, is that these brains are so rare that they cannot figure to any great extent in the common run of those diseases or surgical conditions which they are popularly supposed to accompany and that in many instances the inferences drawn at the autopsy table are incorrect ones based on an improper interpretation of the ordinary appearances of pia arachnoidal collections of fluid, especially where there is also present some widening of the perivascular spaces. It must be remembered, in referring to Kolisko's cases, that the number of autopsies which he has performed is enormous.

Another type occasionally seen is that in which there is general apparent increase in the volume of the brain, diminution in the size of the ventricles, decreased or absent subarachnoid fluid and pronounced flattening of the convolutions; but in none of these cases has there been any increased moisture from the cut surfaces of the hemispheres. On the contrary they have exhibited a rather more than normally dry or glazed appearance (142).

The relations existing between pial effusions and the appearances described as serous meningitis are such as to create confusion. This is chiefly because the common milky patches on the arachnoid are apt to be mistaken for active inflammatory processes, while any free fluid made out in the subarachnoid space is consequently assumed to be serous unless it is slightly cloudy (143).

A critical review of the literature of serous meningitis leads one to the conclusion that a great deal has been written on the subject which will not bear analysis; indeed, such an examination causes one to question whether, taking into account the various possibilities of mistake, the disease can be said to have been positively demonstrated. Thiemich (144) observes that "the term serous meningitis is primarily anatomical and as such is ambiguous," a statement the truth of which becomes increasingly apparent with added study. The instability of the foundations on which the study of the subject of serous meningitis has been made to rest is made apparent when one analyzes the fourteen cases on which Quincke (145) based his clinical observations. The first six cases were entirely clinical lasting up to five months with acute onset, acute course and recovery. No bacteriological work was done. A second group described as "acute cases with a chronic course," comprises three cases. Two of these came to autopsy and showed chronic internal hydrocephalus with tuberculosis elsewhere in the body.

In a third group comprising five cases and described as "chronic," three came to autopsy. The first of these showed a granular ependymitis with hydrocephalus internus and no tubercles. The second showed chronic hydrocephalus, ventricles very wide and filled with clear fluid, a granular ependyma, meningitis, extensive milky clouding of the pia, and slight opacity of the membranes of the cord. There was a large abscess in the pelvis, pyemic abscesses in the lungs, an old endometrit's, and "myelitis (?)." No bacteriological examination was made. In a third case enormous hydrocephalus (800 c.c. of fluid) was present. For the entire group no histological findings are recorded, no bacteriological work was done, and intracranial pressure was measured only five times. Every case of Quincke's, therefore, which came to autopsy, was a case of internal hydrocephalus. It is hard to see on what grounds he based his assumption that in these cases the causative factor was a serous inflammation of the meninges. At the same time Quincke (146) informs us that "he did not find that the fluid in chronic serous meningitis differed from ordinary cerebrospinal fluid. Believing as I do that the spinal fluid is a ventricular secretion (147), I cannot see how this point is demonstrated or how he differentiates his cases from the ordinary form of internal hydrocephalus.

Thiemich, as already noted, looks on the so-called acute hydrocephalus of childhood as a ventricular serous meningitis. He considers that the fluid of "internal" serous meningitis is usually clear but that there are slight changes which indicate inflammation of the chorioid plexus and ependyma (148). Possibly the terms "chorioiditis" and "ependymitis" are more suitable here.

Quincke makes a statement in his article that has a distinct mechanical significance; he says: "In general venous stasis (as in valvular heart disease) we see, in addition to edema of the brain substance, increase in fluid not only in the ventricles but also, and usually in a higher degree, in the subarachnoid space, whereas local venous stasis leads only to transudation into the ventricles with transudation into the brain and compression of the convolutions against the skull."

Edema of the brain substance with increase in the ventricular and subarachnoid fluids is obviously a mechanical impossibility as already pointed out. The second statement implies a closure of the iter or foramina of Magendie.

Boenninghaus' series is equally as unsatisfactory as Quincke's. It is observed that most of his cases can readily be accounted for as either the beginnings of acute purulent or epidemic cerebrospinal meningitis, or as the chronic and partly healed phases of the same conditions.

It is hard to see how the condition can be demonstrated by the histological findings when we consider that the only way one can really prove that a given case of meningitis is serous in character and not some form of leptomeningitis with brain shrinkage, is by the presence of serous exudate.

Thiemich lays great emphasis on the bacterial, microscopical, and chemical examination of the spinal fluid in these cases and it seems safe to say that this is the only satisfactory method in making a diagnosis.

Part 4. The Clinical Significance of Pial Edema.

There are one or two points of importance that require consideration before closing. Of these the first to be considered is the question of the extent to which high intracranial pressure accompanies pial edema.

The simplest method of estimating intracranial pressure post mortem, as has already been stated, is by observation of the tenseness and bulging of the dura just after the skull is opened. Although only a rough guide, this method may be regarded as accurate enough for present purposes. In twenty-eight consecutive cases of pial edema examined, the dura was found tense in ten, indicating that high intracranial pressure coexisted with pial edema in more than one-third of the cases. Proof that the two conditions do not necessarily coexist is furnished by a case of pial edema which was tapped several hours before death by Dr. H. V. Guile, former interne of the Bellevue Hospital staff. The intracranial tension, as measured by him, was normal (150 mm. of H_2O) (149). Certain writers on alcoholism, including some recent ones, have looked on the presence of pial edema as evidence of high intracranial tension; it seems wise in this connection to point out that in many cases symptoms which may have been due to coexistent high intracranial pressure were wrongly ascribed to a "wet-brain" found post mortem.

The second question worthy of attention is: May pial edema be looked on as a cause of symptoms? I have been at some pains to question the various house physicians as to the ante-mortem conditions of the cases on which I performed the head autopsies, particularly those cases in which much edema was present. In Table 5 are the results obtained by this system of inquiry.

I give here a report of all cases as they occurred regardless of whether or not edema was present, but regrouped for convenience of study.

Table 5.—Report of All Cases as They Occurred.

Group 1

Acc. No.	Degree of Edema	Disease	Mental Condition Just Before Death
2,039	XXX	Tuberculous meningitis....	Stupor present.
2,057	XX	Brain tumor	Stupor present; (intracranial tension antemortem was 150 mm. of H_2O).
2,085	XXX	Congenital diplegia	Torpid from birth.
2,111	XX	Uremia	Uremic coma.
2,152	X	Tuberculous meningitis ..	Stupor present.

In this group are placed those cases in which the nature of the lesion is such that it would be difficult to determine the relation of stupor to the coexisting edema. Case 2,057, mentioned above, is of particular interest as suggesting that stupor with brain tumor is not necessarily dependent on high intracranial tension, as the tension in this case is normal.

Group 2—No Pial Edema Present

Acc. No.	Degree of Edema	Disease	Mental Condition Just Before Death
1,907	.....	Chronic pulmonary tuberculosis.	No cerebral symptoms.
1,959	.....	Delirium tremens	Delirious till death.
1,974	.....	Polyserositis	Convulsions.
2,099	.....	Fatty liver; cause of death unknown; infant.	
2,109	.....	Pulmonary tuberculosis...	No pressure or other cerebral symptoms.
2,146	.....	Carcinoma of stomach...	No cerebral symptoms.

The cases in this group have been included merely for the sake of completeness.

Of the following eighteen patients, nine had absolutely no symptoms of cerebral disturbance. Five were irritable or delirious, and four stuporous or comatose. Of the four cases last mentioned, two presented the picture known as "alcoholic wet-brain," a third was profoundly stuporous and a fourth comatose. Although included in this group, it is obvious that coma in a case of perforated gastric ulcer with fatal hemorrhage (Acc. No. 2,133) might readily be due to other factors than those caused by pressure of fluid on the cortex.

Group 3.—Cases Suitable for Study of the Relation of Pial Edema to Symptoms of Stupor or Compression

Acc. No.	Degree of Edema	Disease	Mental Condition Just Before Death
1,879	XX	Cirrhosis of liver.....	No evidence of cerebral disturbance.
1,880	XXX	Interstitial pneumonia ...	No signs of cerebral compression.
1,891	XXX	Act. par. nephritis	Had no cerebral symptoms until just before death, when he became slightly delirious and later sank into a stupor, about which there was nothing characteristic.
1,892	XXX	Epithelioma of esophagus.	Died in delirium.
1,903	XX	Chronic cardiovascular disease.	Was never comatose, nor did he have any mental symptoms beyond the irritableness common to advanced alcoholics.
1,912	XXXX	General arteriosclerosis ...	Characteristic mental symptoms of senility.
1,963	XX	Cardionephritis	No cerebral symptoms.
1,964	XXX	Carcinoma of esophagus..	No cerebral symptoms.
2,052	XXXX	Chronic interstitial nephritis.	Stupor was present.
2,082	XX	General miliary tuberculosis.	No cerebral symptoms.
2,113	XX	Chronic alcoholism; gangrenous cervitis.	Died in delirium.
2,112	XX	Landry's paralysis	Conscious till death.
2,117	X	Chronic glomular nephritis.	No cerebral symptoms.
2,133	X	Perforated gastric ulcer....	Died in coma.
2,147	XXX	Pulmonary tuberculosis; chr. alcoholism.	Irrational, wandering and partly stuporous for some time before death.
2,148	XXX	Pulmonary tuberculosis; chr. alcoholism.	Moderate degree of stupor present.
1,949	XX	Secondary hemorrhage ...	No cerebral symptoms.
2,153	XXX	Emphysema	Rational all the time; died suddenly on attempting to sit up in bed.

On review, there appears in these findings no evidence in support of the idea that pial edema is in itself a producer of symptoms. The symptoms of delirium tremens and "alcoholic wet-brain," it should be remembered in this connection, have been ascribed more properly by others to toxemia. My observations thus have led me to believe that such symptoms as have been recorded are the result of high intracranial pressure or toxemia (CO^2) or other poisons), or both, coexisting with pial edema, but not otherwise related to it.

Conclusion.

My conclusions may be summed up as follows:

1. The collection of fluid in the pia matter is not per se a pathological process but in every instance represents the reciprocal of brain shrinkage.
2. It produces per se no symptoms.
3. Thickenings of the meninges in such cases take place chiefly in the arachnoid and are not to be regarded as representing a true inflammatory process.
4. The brain increases in size up to puberty and diminshes thereafter proportionately to its diminution in weight, as shown by comparison with the tables of brain weights presented.
5. Increase or diminution in size of the brain is not determined by the state of nutrition of the rest of the body.
6. The appearance of "wet-brain" so called, or pial edema of alcoholics, is due to brain shrinkage and has no pathological significance per se.
7. The appearances of pia-arachnoidal effusion are readily mistaken for those of serous meningitis.
8. When a collection of fluid is present in the pia arachnoid, a diagnosis of edema of the brain is open to question and can be made only on the assumption that the brain has been previously in a more shrunken condition.
9. We see no reason to believe that transudation can be a causative factor in the production of cerebral edema.
10. Cases of swollen or edematous brains with dry pias are decidedly uncommon at autopsy and the diagnosis of cerebral edema should be made only when actual increase in the volume of the brain matter can be proved.

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29. Senator: Diseases of the Kidneys, Nothnagel's Encyclopedia, "Am. Ed.," p. 103.
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33. Rokitansky: Lehrbuch der pathologischen Anatomie, 1856, ii, Ed 2.
34. Wunderlich: Die Pathologie and Therapie, 1854, iii, Ed. 2.
35. Leubuscher: Die Pathologie und Therapie der Gehirnkrankheiten, Berlin, 1854.
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53. Verhoogen, R.: La meningite sereuse, "Jour. d. med.," Brux, 1907, 111.
54. Hafslauer: Die bakteriologischen Befunde bei der citehigen und serösen Meningitis mit besonderer Berücksichtigung die bei der Lumbalpunktion, etc., Internat. Contralbl. f. Ohrenh., Leipsic, 1906-1907, i, 65.
55. Blau, A.: A Case of Serous Meningo-Encephalitis with Autopsy Report, Ztschr. f. Ohrenh., 1906, lii, 129.
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57. Riebold, G.: Ueber serosa Meningitis, Deutsch. med. Wchnschr., 1906, xxxii, 1859.
58. Thiemich, M.: Serous Meningitis in Diseases of Children, Pfaundler and Schlossman, Eng. Trans., 1908, iv, 376.
59. Paradis, A.: Ueber Meningitis serosa, B. George, Leipsic, 1906, p. 26.
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61. Williams, L.: Serous Apoplexy, Med. Press and Circular, 1906, lxxxi, 499.
62. Thiemich (Serous Meningitis in Diseases of Children, p. 413) considers that the fluid of internal serous meningitis is usually clear but that there are slight changes which indicate inflammation of the chorioid plexus and ependyma.
63. Boenninghaus: Die Meningitis serösa acuta, p. 93.
64. Thiemich: Serous Meningitis, in Diseases of Children, p. 415.
65. Eighty-two infants were examined; of these eighteen presented pial effusion, that is 21.9 per cent. of the total number of cases.
66. The ages of the thirty-one children mentioned above were as follows: 13 hours, 20 hours, 2½ days, 13 days, 21 days, 1 month (three cases), 6 weeks (two cases), 7 weeks, 2 months (nine cases), 9 weeks, 10 weeks, 3 months (nine cases).

67. A case of marked pial edema in a boy of 15 who died of juvenile paresis was observed subsequently. In this case the brain shrinkage is readily explainable on pathological grounds.

68. The tabulated cases on which these statistics are based were so extensive that it was found impracticable to include them in this article. I shall be very glad to submit them to anyone interested.

69. In forty cases with pial edema the microscopical sections showed no trace of any kidney lesion, while in about the same number of cases there were noted only slight or beginning renal changes. It should be noted that the single case of uremia which we had, showed a slight degree of pial edema. There was no moisture of the cut surface apparent to suggest cerebral edema, no flattening of the convolutions or any other sign characteristic of the brains described by Traube.

70. Cases of serositis not included in this summary.

71. Anton: Wahre Hypertrophie des Gehirns mit Befunden von Thymusdrusen und Nebennieren, Wien. klin. Wchnschr., 1902, xv, 132.

72. Bartel, Julius: Ueber die hypoplastische Konstitution und ihre Bedeutung, Wien. klin. Wchnschr., 1908, xxi, 783.

73. A time of life in which there is a very high death-rate among alcoholics.

74. For a consideration of this subject dealing with meningitis, I would refer the reader to the recent article by W. J. Elser and F. M. Hunton; Studies in Meningitis, Jour. Med. Research, 1909, xx, 371.

75. The blood-vessels lie close to the pia.

76. Myers, V. C.: The Cerebrospinal Fluid in Certain Forms of Insanity with Special Reference to the Content of Potassium, Jour. Biol. Chem., 1909, vi, 115.

77. Dufour, H.: Cytologie du liquide céphalo-rachidien dans un cas de méningite chronique alcoolique, Bull. et. mem. Soc. med. d. hop. de Paris, 1901, xviii, 1035.

78. Halliburton in Kirke's Physiology, Ed. 1908, p. 178.

79. Spinal Fluid Analyses in Alcoholic Wet Brain Cases, loaned by Professor Hastings of Cornell University.

80. Kaufmann, Eduard: Lehrbuch der speziellen pathologischen Anatomie, Berlin, 1904, p. 983.

81. Monro, Alexander: Observations on the Structure and Functions of the Nervous System, Edinburgh, 1783.

82. Kellie, George: Reflections on the Pathology of the Brain. Tr. Med.-Chir. Soc. Edinburgh, 1824, i, 84.

83. Reid: Physiol., Anat. and Path. Researches, xxv.

84. Naunyn, B., and Schreiber, J.: Ueber Gehirndruck, Arch. f. exper. Path. u. Pharmakol., 1881, xiv, 1.

85. Falkenheim, H. and Naunyn, B.: Ueber Hirndruck, Arch. f. exper. Path. u. Pharmakol., 1887, xxii, 261.

86. Horsley, V., and Spencer, W.: Phil. Tr., 1891.

87. Horsley, Sir Victor: The Mode of Death in Cerebral Compression and Its Prevention, Quart. Med. Jour., London, 1894, ii, 305; The Structure and Functions of the Brain and Spinal Cord, P. Blakiston's Son & Co., Phila., 1892.

88. Spencer, W.: The Central Nervous Mechanism of the Respiration, Arris and Gale Lectures, Lancet, London, 1895, xxxi, 532.

89. Roy, C. S., and Sherrington, C. S.: On the Regulation of the Blood Supply of the Brain, Jour. Physiol., 1890, xi, 85.

90. Hill, Leonard: The Physiology and Pathology of the Cerebral Circulation, London, 1896, Churchill.

91. Kocher, T.: Hirnerschutterung, etc. Specielle Pathologie und Therapie in Nothnagel, Wien, 1901, Ed. 2, Part 3, ix.

92. Cushing, H.: Some Experimental Clinical Observations Concerning States of Increased Intracranial Tension (Mutter Lecture), Am. Jour. Med. Sc., 1902, cxxiv, 375; The Blood Pressure Reaction of Acute Cerebral Compression, etc., Am. Jour. Med. Sc., 1903, cxxv, 1017; A Discussion of Some Remote Effects of Cranial Injuries, etc., New York Med. Jour., 1907, lxxxv, 97, 61, 208.

93. Burrows, George: Disorders of the Cerebral Circulation and on the Connection Between Affections of the Brain and Diseases of the Heart, Philadelphia, 1848, Lea and Blanchard.

94. Donders, F. C.: Nederl. Lancet, 1850.

95. Reynolds and Bastian: Congestion of the Brain, in Reynolds' System of Medicine, 1879.

96. Hill, Leonard: The Physiology and Pathology of Cerebral Circulation, p. 36.

97. Schafer, E. A.: In Quain's Anatomy; Spinal Cord and Brain, Part 1, p. 3, Longmans, Green and Co., London, 1893.

98. The suggestion that the theca vertebralis plays a part during life in the accommodation of excessive amounts of spinal fluid and the production of high intracranial pressure will doubtless arouse opposition among those who have demonstrated vascular relationship to pressure phenomena. I do not, however, disagree with Dr. Ferrier's statement that under normal conditions the ebb and flow between the cranial and spinal cavities is so small as to be practically a negligible quantity, but I desire to point out that no experimental work so far done is of much value in helping us to reach conclusions in the present work: largely because of the fact that none of these experiments has been arranged so as to show what mmechanical changes take place when resorption of spinal fluid is prevented and when the high pressure is caused by an excessive amount of the fluid itself. My suggestion that distention of the theca is a factor is based on a study of the behavior of fluids within the craniospinal canals of cadavers. The distensibility of the sac under these circumstances has long ago been demonstrated, but Dean (Jour. Path. and Bacteriol., 1893, i, 28) and others have been inclined to doubt whether such a process could take place in living subjects.

It is of course unlikely that such a process would take place unless there were some hindrance to resorption.

99. Water, which is relatively incompressible, has increased in weight only about 0.03 pound to the cubic foot at a depth of 350 feet. Treatise on Hydraulics, Merriman, 1906, p. 11.

100. The subject of the effects of pressure on the vascular structures within the skull has received so much attention of late that elaboration in this paper would seem superfluous. The following contributions are especially important: Ferrier, David: The Harveian Oration on the Heart and Nervous System, Lancet, London, 1902, ii, 1099; Cushing, H.: ibid.

101. Parrot: Bull. Soc. d' Anthropol. de Paris, 1887, le Mars.

102. Pfister, H.: Ueber das Gewicht des Gehirns und einzelner Hirntheile beim Saugling und alteren Kinde, Neurol. Centralbl., 1903, xxii, 562.

103. Marchand: Ueber das Hirngewicht des Menschen, Abhandl. d. math.-phys. Classe d. kgl. Sachsischen Gesellsch. d. Wissensch., 1892, xxvii, 437.

104. Vierordt, H.: Anat. Physiol. u. Physikal, Datun und Tabellen, 1906, Jena.

105. Boyd: Phil. Tr. Roy. Soc. London, 1861, cli, 242.
106. Meynert, Th.: Das Gesammtgewicht und die Theilgewichte des Gehirnes in ihren Beziehungen zum Geschlecte, dem Lebensalter und dem irrsin, untersucht nacht einer neuen Wagungsmethode an den Gehirnen der in der Wiener Irrenanstalt im Jahre 1866 Verstorbenen, Vrtljschr. f. Psychiat., 1867, ii, 125.
107. Handmann, Ernst: Ueber das Hirngewicht des Menschen, etc., Arch. f. Anat. u. Entwcklngsgesch., 1906, p. 1.
108. Von Bischoff, T. L. W.: Das Hirngewicht des Menschen (von P. Neusser) Bonn, 1880.
109. Quoted from Quain's Anatomy, iii, Part 1, p. 178.
110. Blakeman, J.: A Study of the Biometric Constants of English Brain Weights and Their Relationships to External Physical Measurements, Biometrika, 1905, iv, 124.
111. Blakeman, J.: Biometrika, 1905, iv, 138. He found no shrinkage in the head diameters of criminals, but states that the subject has not been fully worked out for the general population.
112. Clapham, C.: The Weight of the Brain in the Insane, West Riding Asyl. Med. Rep., iii, vi.
113. Crichton-Browne, J.: The Weight of the Brain and Its Component Parts in the Insane, Brain, 1879, i, 504; ii, 42.
114. Mercier, C. A.: The Weight of the Brain in the Insane with Reference to Hemispheres, Lobes, Brain-Stem and Cerebellum, Jour. Ment. Sc., 1891, xxxvii, 207.
115. Donaldson, H. H.: Growth of the Brain, 1895, pp. 137-140.
116. Kaufmann, E.: Spezielle Pathologische Anatomie, Ed. 5, Berlin, 1909, p. 1094.
117. My figures already given under "Nutrition" in Part 1 have shown an absence of relationship between pial edema and wasting of the rest of the body.

118. The shrinkage in the brains of chronic alcoholics may be explained by the noteworthy changes in the ganglion cells of such cases as taught and demonstrated by Dr. Ira Van Gieson many years ago.
119. Quoted by A. E. Russell, p. 1095.
120. Gowers, William R.: Abstract of a Lecture on The Nervous System in Old Age, Polyclinic, London, 1907, xi, 131.
121. Mott: Arch. Neurol., London, i, 499.
122. Personal Communication.
123. Huguenin: Handbuch der speziellen Pathologie und Therapie von v. Ziemssen, Suppl. Vol., 1878, p. 6.
124. The following descriptions taken from autopsy report No. 2,402, are interesting since they show that transudation does not necessarily take place within the skull as a result of thrombosis of the veins of the neck.

"Head: The longitudinal sinus is free; there is marked pial edema and a moderate amount of fluid at the base. The cerebral vessels are everywhere thin and normal. The pial veins over both superior convexities are normal with the exception to be noted below. The temporosphenoidal and occipital veins on the surface of the right side are the seat of firm, blackish coagula. On removing the brain, a large vein with its branches filled with blackish clot is found torn and lying on the superior surface of the petrous portion of the temporal bone. This vein evidently formed a communication between the above-named veins and the lateral sinus. The lateral sinus and the bulb on the right side is the seat of a dark coagulum which is markedly adherent to the wall of the vein. The right middle ear contains an excessive amount of clear fluid. The right mastoid cells appear to be mostly closed, the bone being extremely firm and discolored yellowish. The left middle ear and mastoid normal. Cross-section of the brain reveals no gross lesions. There is no edema of the brain substance. The lateral ventricles were not distended and contained a normal amount of fluid.

"Blood-Vessels: The inferior vena cava is normal. The superior vena cava, the right innominate, right and left internal jugular and left external jugular veins are markedly thickened and the seat of an old firmly adherent and partially organized thrombus, which is paler at its cardiac end than toward the head."

125. Leyden, E.: Beiträge und Untersuchungen zur Physiologie und Pathologie des Gehirns, Arch. f. Path. Anat., 1866, xxxvii, 519.
126. Duret, H.: Etude expérimentales et cliniques sur les traumatismes cérébraux, Paris, 1878, p. 183.
127. Cybulski, N.: Zur Frage des Gehirndruckes, Centralbl. f. Physiol., 1891, iv, 834.
128. Hill, L.: The Physiology and Pathology of Cerebral Circulation, p. 168.
129. Cannon: Am. Jour. Physiol., 1901, vi, 101, 102.
130. Budgett, S. P.: The Similarity of Structural Changes Produced by Lack of Oxygen and Certain Poisons, Am. Jour. Physiol., 1898, i, 210.
131. Loeb: Arch. f. d. ges. Physiol., 1898, lxxi, 47.
132. Cannon: Am. Jour. Physiol., 1901, vi, 114, 115.
133. Cannon: Am. Jour. Physiol., 1901, vi, 119.
134. Huguenin has discussed this matter rather carefully. It seems curious that some writers have failed to realize that the cranial subdural space is distinct from the subarachnoid space and its spinal prolongation, for, owing to the close apposition of the membranes, at the foramen magnum, there is no subdural space in the spinal canal. To illustrate my meaning, bloody spinal fluid has been considered to be an evidence of middle meningeal hemorrhage, whereas the presence of blood in this fluid in such cases is only an indication of rupture of the dura and subarachnoid membrane or cerebral laceration. In suppurative internal pachymeningitis the cerebrospinal fluid is clear unless there has been an extension of the process to the pia.
135. Fischer, Martin H.: The Nature and the Cause of Edema, The Journal A. M. A., 1908, li, 830.
136. This theory of colloidal absorption is not generally regarded as proved. It is well known, however, that myelin substances are capable of absorbing large quantities of water.
137. Unless we allow the brains in question to have been markedly shrunken beforehand and admit that they have become swollen since. In such cases we can at least be sure that the edema itself could have played no part in the production of pressure symptoms.
138. On page 488 Cushing describes the operation at this stage as follows: "The dura was exceedingly tense and owing to the marked degree of cerebral protusion that followed the first incision, it was opened with considerable hesitation in the absence of coincident lumbar punctures. It was accomplished, however, without injury to the pia arachnoid. The subdural space contained no free fluid. The arachnoid on the other hand was markedly distended with fluid which escaped after pricking the membrane in a number of non-vascular spaces where it bridged the exposed sulci. Not only was there a superabundance of fluid in the arachnoid space but the brain itself appeared

soggy and wet." This picture it seems is hardly compatible with our conception of the swollen edematous brains described by Traube unless we accept the paradox that in this instance both brain volume and subarachnoid fluid contents were simultaneously increased. The mechanical complications arising as the result of a subsequent cerebral hemorrhage in the right hemisphere (see page 494 autopsy report of same article) certainly renders the case of little value from the standpoint of the study of cerebral edema.

139. Phelps: Traumatic Injuries of the Brain and Its Membranes, vi, 536.

140. Cannon: Am. Jour. Physiol., 1901, vi, 97.

141. Dr. Norris tells me he has never observed this condition, although he has for four years been on the watch for it.

142. A case of this sort is included in our tables of cranial weights and volumes, note Group II, Accession No. 2,099, Table 5.

143. In such cases a careful bacteriological examination must be made to eliminate a beginning inflammatory process, especially in pneumonia.

144. Thiemich, Serous Meningitis in Diseases of Children, p. 413.

145. Quincke: Samml. klin. Vortr., pp. 656-668.

146. Quincke: Samml. klin. Vortr., p. 678.

147. The reader is referred to Brodie's Harvey Lecture, New York, 1909, on Renal Activity (the chorioid plexus being supposed to functionate as do the glomeruli).

148. For composition of the fluid in hydrocephalus, see under spinal fluid, Text-Book of Physiological Chemistry, Hammarsten Ed., 1908, p. 264.

149. See Accession No. 2,057, in Table 5.

MISTAKES IN THE DIAGNOSIS OF TYPHOID FEVER.*

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The average patient expects a physician to make a correct diagnosis of his ailment at the first professional visit. The younger the physician the more insistent is this demand, and an honest acknowledgment by the doctor of his inability to comply with the demand at this time often results in his involuntary retirement from the case. An older physician may be pardoned for his honesty and permitted to continue his observations especially if he is one of the diplomatic brethren whose clients are frequently told that they are "threatened" with a serious disease. When the "threatened" calamity does not subsequently develop, its defeat is then attributed to the superior methods of treatment adopted. But typhoid fever, at its outset, is so protean in its manifestations, and shows so many variations in its symptoms and course, and is simulated by so many different febrile and inflammatory affections that its immediate diagnosis at the bedside may be impossible. It is only by keeping its possibility always in mind that the chances of overlooking it, of mistaking it for other diseases, or of mistaking other diseases for it, will be minimized. The typical text-book case will usually be promptly recognized, but the typical case is usually found in the text-books rather than at the bedside. A recital of some of the mistakes, my own as well as those of others, that I have encountered, may serve to fasten the differential points in your memory as they have in mine. Many of these mistakes would not occur in a well-equipped hospital with excellent laboratory facilities, but we have not yet become sufficiently paternalized in this country to be able to afford to relinquish all our typhoid patients to the care of the well-endowed hospitals and laboratories. The careful clinical observations of a qualified practitioner, corroborated by the simpler laboratory blood tests and blood counts which every recent graduate is taught to make, will steer us aright in most cases. It is only their neglect which serves to emphasize the truth of the old remark, that nine-tenths of the mistakes in differential diagnosis are made by not looking, and only one-tenth by not knowing. Blood cultures, which need an elaborate laboratory equipment, are rarely necessary. The Widal reaction, available after the seventh day, and the differential leucocyte count,

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available on the first day, will furnish all we ought to ask—data to disprove the existence of most of the simulating diseases. Given the general picture of a febrile infectious disease, there are only six acute infections to which leucocytosis is absent, to wit: malaria, typhoid fever, influenza, measles, mumps and leprosy. To these may be added most cases of uncomplicated tuberculosis, except the meningeal form. Let us consider some differential points.

1. Tuberculosis.—Acute miliary tuberculosis may present all the symptoms and signs of typhoid fever, even including the rose rash. But the respirations will be more frequent, the pulse will be proportionately rapid instead of proportionately slow and dicrotic, cyanosis will be more apt to appear, the abdomen may be flat instead of tympanitic, the temperature will be irregular instead of definite in type, phonophobia and photophobia will be notable, petechiae may be present instead of rose spots. Widal reaction will be wanting, and a tub bath will produce a greater drop in the temperature than 2 degrees F. A case in Bellevue Hospital at present also shows tubercles in the choroid, but this is very rare. Lumbar puncture may show tubercle bacilli in the spinal fluid.

2. Malaria.—In the past the term malaria has covered a multitude of diagnostic sins, but the present generation hears less of "typhomalarial fever" or "bilious remittent fever" than we did thirty years ago when hematology was an infant science. Still, estivoautumnal fever, with its continued temperature, its septic aspect, and the difficulty of finding the crescents in the peripheral blood may keep us guessing for some days. At St. Vincent's we get many seafaring men from Southern ports with the estivoautumnal type of fever and are on the watch for it. But it may arise also in our latitude. I have seen cases that originated on the shores of Jamaica Bay, in the Borough of Queens.

3. Typhus Fever.—The sporadic cases of typhus fever that we see in this vicinity are of the attenuated type, so well described recently before this society by one of your members. It seems to be more prevalent among certain of your neighbors than with us across the Williamsburgh Bridge. I have seen but two cases in Bellevue in the last two years and none in St. Vincent's. It is easily recognized when once known.

4. Septicemia.—Originating after labor or after abortion, this condition is often miscalled "typhoid fever," especially by that class of practitioners who have "never lost a woman after confinement." Leucocytosis and an increase in the polymorphonuclears will assign the proper label to this condition. A fatal case of gonorrheal septicemia with ulcerative endocarditis, occurring in a young man with old rheumatic simple endocarditis, was sent into our wards at Bellevue with a diagnosis of typhoid fever, but leucocytosis suggested and blood culture proved the actual condition. An additional reason, if any were needed, why young people with chronic endocarditis should avoid tacking on an acute gonorrheal infection.

5. Endocarditis.—As in the supervention of gonorrhea, the addition of typhoid bacilli to the circulating blood of a patient with old endocarditis renders the liability to ulcerative lesions of the heart lining very grave. An undamaged endocardium usualy resists their invasion.

6. Influenza.—Many cases of influenza are so typhoidal in their aspect at the onset that only the subsidence of the influenzal symptoms in a few days dispels our anxiety. Some cases may require a blood culture to decide.

7. Febricula.—Many of the cases of short continued fever formerly classified under this head and some of the so-called "gastric fever" cases would now be demonstrated by the Widal reaction to be mild forms of typhoid.

8. Intoxication.—The various autointoxications which for want of more definite knowledge we regard as ptomaine poisoning can usually be differentiated

by their suddenness of onset, the violence of their initial symptoms and their reference to an obvious dietetic indiscretion.

9. **Osteomyelitis.**—A case seen by me was that of a young man who, suffering from a crop of boils, worked at a hand drill all the week, pitched baseball on Sunday, and was taken ill with chill, sweats, high fever, and pain referred to the shaft of the humerus. He was treated four days for malaria, three days for rheumatic fever, three days for typhoid fever, this by four different physicians. The leucocyte count was 26,000 with 89 per cent. polymorphonuclears and trephining the shaft of the bone at St. Vincent's Hospital confirmed my diagnosis. This case had almost become one of the tragedies resulting from "not looking." Osteomyelitis has been called "bone furunculosis." It is one of the diseases we should always think of in childhood and adolescence.

10. **The Acute Exanthemata.**—Measles, scarlet fever, and variola will soon clear up their own diagnosis and need no description here.

11. **Trichinosis.**—Cases of trichinosis are frequently sent into hospitals with a diagnosis of typhoid fever. Leucocytosis with abnormal eosinophilia will point the way to, and excising a piece of muscle will establish, the correct diagnosis.

12. **Syphilis.**—Secondary syphilitic fever with macular eruption will not be mistaken when associated with sore throat and adenopathy. The absence of the Widal and the presence of the Wassermann reaction will decide the doubtful cases.

13. **Cerebrospinal Fever.**—Leucocytosis and the results of lumbar puncture have recently placed the diagnosis of this condition on much firmer ground. In my earlier days mistakes were common. The same diagnostic procedures will be of much service in the recognition of other forms of meningitis and also in a variety of diseases with nervous manifestations in which fever with more or less stupor are prominent features.

14. **Pulmonary Conditions.**—The initial bronchitis of typhoid fever may be so severe as to concentrate our attention on that condition. Or typhoid may set in with an unmistakable attack of acute lobar pneumonia which may entirely overshadow, or even obliterate, the usual low leucocyte count and slow pulse of typhoid. No amount of care will save us from this mistake, if it can justly be so called. The term pneumotyphoid should not be used for these cases.

15. **Abdominal Conditions.**—An Italian suffering from typhoid fever was admitted to my service in Bellevue who was seized in the middle of the night with violent gastroenteritis after a supper into which green peppers had largely entered. He had been treated for five days by a physician of his own nationality for appendicitis. This was a pardonable error with the given history of sudden onset and violent symptoms. The first typhoid patient of my own upon whom I had mistakenly advised an operation for appendicitis caused me considerable chagrin, although I had only sustained the diagnosis of a man of sufficient experience to be chief of clinic at a college dispensary, and we had both been sustained by the operating surgeon. Study of the literature lessened my shame, when I found that the same error had been often made by the best surgeons. Richardson of Boston emphasizes the most important differential point, to wit: Muscular rigidity is wanting.

16. **Renal Conditions.**—A young policeman came to my office one evening complaining of headache, dimness of vision, and scanty urine. I had incised a

suppurative tonsilitis for him ten days previously. His urine presented the characteristic physical, chemical, and microscopical evidences of acute desquamative nephritis, and he was sent to Bellevue with that diagnosis. He afterward ran the typical course of typhoid fever. This was one of the so-called nephrotyphoid cases.

17. Varieties of Typhoid.—As stated at the outset, age cannot wither nor custom stale the infinite variety of typhoid, and he who studies his typhoid cases attentively will not only acquire a fund of knowledge regarding the working of the acute infections in general, but also a knowledge of the sanitary intelligence of the community in which he resides. Cases may be classified as the malignant, which are usually fatal from cardiac degeneration, severe toxemia, or intestinal hemorrhage and perforation; the mild, which are only dangerous in so far as they may be the means of communicating the disease to others; the ambulatory, which are dangerous to themselves because of their marked liability to hemorrhage and perforation; the hemorrhagic, by which is meant those in which bleeding occurs from various parts of the body, and of which the majority of cases die; the afebrile, which I have never seen; the obese, who, as a rule, do badly.

18. Paratyphoid Fever.—The diagnosis of this condition is made only in the laboratory. Symptomatically, diagnostically, or therapeutically, it does not differ from ordinary typhoid, and it need not cause the working practitioner any more doubt or delay than does the particular organism producing a felon on the finger, whose quietus he knows that he can make with a bare bodkin, without calling in a laboratory man to identify the germ.

HOSPITALS AND TYPHOID CARRIERS.*

JOHN W. BRANNAN, M.D.

During the last two years an increasing amount of attention has been called to the danger to the public health of typhoid carriers, and to the importance of their recognition and control. In August of last year, Meakins (2), of Montreal, in an exhaustive article on the subject, urged that in our general hospitals, where at least 50 per cent. of the cases of typhoid fever are treated, every case of the disease should be examined bacteriologically during convalescence, and should be kept in the hospital or under strict observation and be treated by homologous vaccines until the excreta were free of typhoid bacilli. Meakins further advised that patients under the care of physicians in private practice should be watched and examined from time to time by officials of the board of health. Impressed by the suggestion of the Canadian writer, I requested Dr. Norris, the director of laboratories in Bellevue and its allied hospitals, to have the feces and urine of all cases of typhoid fever examined during convalescence. This work was begun in September, 1911, and has continued up to the present time. One hundred and nineteen cases in all have been examined, and of these, 15, or 12.6 per cent., had typhoid bacilli in either the feces or the urine or in both. An average of two examinations was made in each case. Forty-eight cases were also examined during the active stage, with a positive result in 10, or in 20.83 per cent. From these figures it appears that

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* Extracted from the American Journal of the Medical Sciences, September, 1912, No. 3, Vol. cxliv, p. 347.
Read at the annual meeting of the Association of the American Physicians, May 15, 1912.

about 1 case in 5 gave positive findings during the active stage of the disease, but only in 8 cases by the time they had arrived at convalescence. All patients with the exception of two were bacteriologically free before they left the hospital. These two patients were discharged inadvertently. Their names and addresses and occupations were sent to the Board of Health, though not as promptly as might be wished, as the mistake was not discovered for some time. Our own social service department is also endeavoring to trace them in order that we may make further examinations, if possible, and determine if they are still carriers. At present no typhoid patient is discharged from any of the hospitals in the department except with the approval of the general medical superintendent. We have found no great difficulty in holding patients until they are presumably bacteria free, at least two successive negative examinations being required in every case.

It may be urged that patients discharged as bacteria free after only two examinations with negative results are still potential carriers and would prove to be actually such on further examination. It is believed, however, that this measure of precaution will serve to arrest the great majority. As the work progresses and our methods improve with experience, we shall no doubt be able to increase the number of examinations, at least in those cases that have given positive results at some time in the course of the disease. Dr. Norris is trying to devise some method of securing the bile, which may enable us to arrive at more exact conclusions. Dr. William Hallock Park has also some such procedure in view.

There is one measure at least that we can take to guard the public health against infection by typhoid carriers, either actual or potential, and that is to instruct them in the simple rules of cleanliness. This we should do with all cases of typhoid fever when they leave the hospital or pass from our observation in private practice, but particularly should we do this in the case of those who have shown themselves to be at least transitory carriers. These who are engaged in the preparation or distribution of food should be doubly warned in this regard. We are now preparing a leaflet of instructions to be given to each patient on discharge from the hospital. It would be well, of course, to prevent such persons from following occupations involving the handling of food or drink; but this in many instances would probably be impossible, particularly in this country. At all events, this is the province of the Board of Health, not of the hospital. It is for us to instruct the patient and give notice to the health authorities, that they may take the necessary measures to protect the public. It may be of interest to note the proportion of those engaged in occupations in which they were liable to infect others. In the 140 different individuals who form the subject of this study there were 14 housewives, 3 nurses, 3 waiters, 3 cooks, 1 orderly, 1 butler, and 1 baker—25 in all. One has but to read this list to see how impossible it would be to compel any considerable number to change their occupation. Hence the importance of instructing them both orally and in writing how to avoid conveying infection.

Regulations for the control of typhoid carriers have been enforced in some countries of Europe for a number of years. In the southwestern provinces of Germany there is probably no case of typhoid fever released from observation until the bacteriological examination of the feces and urine has proved negative. In England also typhoid carriers receive a great deal of attention, especially in the English army. After treatment for a period of three months, if it is found that the soldiers are still carriers, they are given the opportunity of entering the hospital or of being discharged. In case of discharge, notification is sent to the medical officer of health of the district in which the soldier is

going to reside. In the service in India two convalescent depots were established several years ago, to which the convalescents of certain military stations are sent. After the opening of these depots, namely, Wellington and Naini Tal, it was found that the admissions of typhoid fever from all stations which sent their convalescents there show a reduction of 9 per cent., in 1908, from the figures for the previous year, whereas the remaining stations show an increase of 26.6 per cent.

In the endeavor to sterilize typhoid carriers various methods of treatment have been tried, such as the use of lactic acid bacilli, acidifying the urine, the administration of antiseptics, the use of the x-rays and the employment of vaccine. The surgeons (3) of the English army report the following results from the use of these five different means of treatment: (1) Lactic acid bacilli cause only a temporary disappearance of the typhoid bacilli. (2) Acidifying the urine fails to cure typhoid bacilluria. (3) The administration of antiseptics invariably brings about a decided diminution in the number of bacilli both in fecal and in urinary carriers. This effect is much more marked when the maximum contact of the antiseptics with the bacilli is obtained by combining the treatment with low diet and aperients in the case of "fecal," and with diuretics in the case of "urinary" carriers. (4) The use of x-rays in the case of gall-bladder infection seems to have definite beneficial results. (5) Vaccines, like the lactic acid bacilli, cause only temporary disappearance of typhoid bacilli. It is suggested that the treatment by vaccines might have a better chance of success if combined with diuretics in the case of "urinary" carriers, and with x-ray treatment in gall-bladder cases. Sir Almroth Wright has pointed out that a vaccine is more likely to be efficient when the local conditions are so altered as to permit the fullest possible contact between the bacteriolytic products in the blood and the bacteria involved.

Other observers have had better results with vaccines than the English army surgeons. Some have reported permanent cures in cases of long standing, notably Meakins (4) in 2 cases described in detail, 1 of chronic bacilluria of twelve years' duration, the other one of chronic suppurative cholecystitis with gallstones of eight years' duration. At Bellevue we have as yet to do only with transitory carriers, but Dr. Norris is prepared to use autogenous vaccination when occasion arises.

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SOME EXPERIENCES WITH ANTI-TYPHOID INOCULATION.*

JOHN W. BRANNAN, M.D.

At the annual meeting of the Association of American Physicians at Atlantic City in May of this year, Dr. Leslie H. Spooner (1) of Boston related his three years' experience with antityphoid inoculation of the nurses in a number of hospitals in Massachusetts. Spooner was led to employ this prophylactic measure because a study of the records of these hospitals had shown a typhoid morbidity rate among the nurses very much in excess of that in the population at large. In the Massachusetts General Hospital it appeared that during the ten years previous to 1909, from two to six of the nurses had come down annually with typhoid fever. Inoculation was, therefore, offered to the members

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of the training schools and was promptly accepted. As a result the incidence of typhoid has practically ceased among the nurses of the hospitals which have adopted the procedure. The reactions following the inoculations varied in severity but were, as a rule, very slight or moderate in character.

After hearing Dr. Spooner's paper I returned to New York with the intention of having the nurses inoculated in the hospitals with which I am connected, Bellevue, Gouverneur, Harlem and Fordham. I had not expected to make the inoculations myself, but there seemed at the time to be no one else to do it. I began the work in June, having been first inoculated myself by Dr. Norris, the director of pathological laboratories of the hospital. He used the vaccine of the United States Army, giving as the first dose ½ c.c., containing 500 million dead bacilli, followed at intervals of ten days with a second and a third dose of 1 c.c. each. After each inoculation the arm at the point of injection was somewhat painful, tender, and swollen for 36 to 48 hours, the tenderness and swelling extending up and down the arm for a few inches. The general reaction consisted of a fairly severe headache, pain in the back, and some malaise, these symptoms lasting for about 24 to 36 hours. After I had received the second inoculation I proceeded to treat the nurses, using vaccine furnished by the Board of Health, of the same strength as that of the army, and in the course of the summer I inoculated some 200 nurses in the hospitals, and also about 50 members of the house staffs, as well as a few private patients, the latter being all men. The inoculations were all given on the outer surface of the left upper arm at the insertion of the deltoid muscle. The skin was sterilized with the tincture of iodine and the injections were made subcutaneously, not into the muscle. There were very few severe reactions, these being more marked among the men than among the women. Not more than three nurses asked to be relieved from duty for a day, whereas several of the internes were laid up for as much as two days at a time with temperatures ranging as high as 102 degrees, with rapid pulse, nausea, vomiting, severe headache, and pain in the back. One of the doctors had a temperature of 103 degrees, following each of two doses, both of them only ½ c.c. He had also what he described as a "splitting" headache, lasting for 36 hours, accompanied with great prostration and pain in the abdomen. Since the second inoculation (he has not wished to take a third) he declares himself as feeling better than ever before in his life. There had been very little typhoid among the nurses in Bellevue or any of the other hospitals, so that there seemed to be no particular reason for giving the prophylactic inoculation, but I urged that it be done, not only to protect the nurses while on duty in the hospital, but as a general measure of precaution, especially as many of them were about to take their vacations in the country, where they would be exposed to infection from sources beyond their control. No compulsion was exercised, but the nurses were advised that it would be to their advantage to undergo the inoculation. The great majority of the pupil nurses responded quite willingly, but a number of the graduates, who came for short periods of post-graduate instruction and experience, begged to be excused.

As I had heard of the occasional lighting up of latent diseases, such as arthritis, I inquired particularly as to whether anything of the kind occurred in these cases, but so far as I could learn nothing of the sort happened. It may also be of interest to know that the reaction was the same in the cases that had had typhoid as among those who gave no history of the disease. When some of the nurses and some of the doctors suggested that, as they had had typhoid fever, perhaps it was not necessary for them to be inoculated, I replied that not only did that not imply that they could not have the disease again, but also that from a scientific standpoint it would be interesting for them to receive

the inoculation as "controls." They were not particularly impressed with my suggestion, which did not seem to appeal to their scientific spirit, but they accepted the treatment just the same.

It is unfortunate that we must use the word "vaccine," as it gives rise to apprenhension as well as misapprehension among the nurses. It calls to mind their experience with vaccination against smallpox, and they are much relieved when they learn that the operation consists of a simple hypodermic injection and that the amount of fluid injected is small. In the army, Russell (2) employs the term "typhoid prophylactic," which has at least the advantage of not giving a wrong impression of the procedure itself.

Many of the nurses while undergoing inoculation were in close attendance on cases of typhoid fever in the wards, and Wright's "negative phase" more than once passed through my mind and the fear that I might be exciting an increased susceptibility to the disease. Nothing happened, however, and the nurses are now protected. It is to be hoped, as claimed by Richardson (3) and Spooner, that the danger of the negative phase of immunity said to follow bacterial inoculation has no substantial basis in fact, otherwise we might hesitate to make use of the procedure at a time when it would be of the most service, that is, in the presence of a local epidemic of the disease.

Some of the private patients who came to me for inoculation did so because they were about to go abroad, and feared that they might be infected by food or drink in the course of their travels. This argument is rather amusing when we consider that typhoid fever is much more prevalent with us than it is in European countries. A recent paper by Dr. Allen C. McLaughlin (4) of the United States Public Health and Marine Hospital Service shows that in 1910 in some 25 European cities there were 6.5 deaths in 100,000 population, whereas in this country in an equal number of cities of the same character there were 25 deaths in the same number of population. In other words, there is four times as much typhoid fever in the cities of the United States as in those of Europe, and as we know there is more typhoid in the smaller towns and in the country districts than in the large cities, it would seem to be much more reasonable for those who are about to spend their vacations in the country regions in the United States to be protected by inoculation than for those who are going abroad.

Many persons have wished to know the duration of immunity after inoculation, and I have usually replied "two or three years." Colonel Firth of the British Army, quoted by Major Russell (5), concludes from his experience in India that immunity begins to diminsh in about two and one-half years. In our own army, according to Russell, it is the present practice to revaccinate against both smallpox and typhoid at the beginning of each three-year period of enlistment. Martha Wollstein (6), in a recent study on "The Duration of Immune Bodies in the Blood after Antityphoid Inoculation," found that these bodies reached their height within two months after the first inoculation and then fell rapidly within the next two months. Of nineteen cases under observation, the blood of fifteen was negative after thirteen months. Although, as Dr. Wollstein states, experience has proved that clinical immunity cannot be determined absolutely by the measure of immune bodies in the blood, she nevertheless believes that reinoculation, with typhoid vaccine within a year is indicated when exposure to typhoid fever seems imminent. Russell, while noting the disappearance of agglutinins in little more than a year, adds, however, that they are present as long after inoculation as after typhoid fever, which gives, as a rule, protection for life."

I should like to know if any one present has had any experience with the

employment of sensitized bacilli instead of dead bacilli. In a recent number of the "New York Medical Journal" (7) reference is made to the method of Vesredka, in which 500 to 750 million sensitized living bacilli are given as a first dose, to be followed by a second dose of double the quantity some seven to nine days later. It is stated that there is no general reaction and only an insignificant local reaction following each injection. The paper to which the "Medical Journal" refers is by Alcock (8), and is published in the "Lancet" of August 24. It is also stated that all authors agree that the vaccination by living microorganisms is more effective than when the dead organisms are used. If this be true, and the reaction is less severe, it would seem to be well for us to make use of this newer method, provided it can be proved to be free from the danger of conveying typhoid fever.

Since the above paper was read the records of the Bellevue Training School for the last twenty years have been carefully gone over by Dr. Robert J. Carlisle, the attending physican to the school, and Miss Brink, the superintendent in immediate charge of the school, and they were agreeably surprised to find that during this long period there had been but eleven cases of typhoid fever among the nurses in the hospital. It would be difficult, perhaps impossible, to ascertain the number of nurses serving in the hospital during all these years, but it is undoubtedly very large. In addition to 525 nurses who completed their course of training, there must be counted the probationers who failed to qualify as pupils in the Bellevue school, and the hundreds of affiliating nurses and graduates of other hospitals who came to Bellevue for post-graduate instruction and experience.

Another note of interest must be added. Early in November a pupil nurse—now happily convalescent—contracted typhoid fever in the hospital She was one of seven who had refused inoculation. On the other hand, of eighty-three who accepted the typhoid prophylactic not one has acquired the disease, though they were all more or less exposed to infecton throughout the summer and early autumn. The occurrence of this case has not unnaturally had a marked effect in influencing the other nurses to undergo inoculation.

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THE HIGH CALORY DIET IN TYPHOID FEVER: A STUDY OF ONE HUNDRED AND ELEVEN CASES.*

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In 1909 Dr. P. A. Shaffer and the author (1) published the results of an investigation of the protein metabolism in typhoid fever. This investigation proved that by the use of diets of high caloric value, especially when rich in carbohydrate, it was possible to diminish, and, if the supply of carbohydrate was suf-

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(From the Second Medical Division of Bellevue Hospital and the Department of Applied Pharmacology, Cornell University Medical College.)

ficient, to prevent the febrile loss of body protein, which on all previous diets had been so characteristic of this disease.

On the basis of this investigation a new diet was arranged for the treatment of typhoid fever. But no attempt was made in the paper referred to to discuss the therapeutic value of the diet.

A brief clinical paper (2) upon the subject was also published in 1909, but, in this paper, it was not possible to consider in sufficient detail either the diet or its administration.

In the four years which have elapsed since the investigation was begun, the high calory diet has been employed in the treatment of 111 cases of typhoid fever in the wards of the Second Medical Division of Bellevue Hospital. The experience derived from these cases forms the basis of the present paper. While it is my purpose to consider mainly the clinical application of the high calory diet, it will be necessary to refer from time to time to the scientific aspects of the subject.

The Daily Food Requirement.—Shaffer and Coleman calculated theoretically the minimum daily food requirement of the average adult typhoid fever patient to be about 40 calories per kilogram of body weight, or approximately 3,000 calories for a patient weighing 150 pounds, but their investigation showed that, in the cases studied, a mixed diet furnishing this amount of energy was not sufficient to establish nitrogen equilibrium. The best results in the maintenance of nitrogen equilibrium were obtained with diets furnishing from 60 to 80 calories per kilogram per day, or from 4,000 to 5,500 total calories. The optimum requirement, however, varied greatly in different cases and at different stages of the disease, but was always greater than the calculated minimum.

Grafe (3) found recently, in a study of the total metabolism of 12 cases of typhoid fever in the fasting state, that the heat production amounted only once to 40 calories per kilogram (the total in this instance being 1,740), and never exceeded 2,798 calories a day.

While the discrepancy in these results is accentuated by the fact that Grafe's patients were fasting, it cannot, with our present knowledge, be explained. But, on the basis of clinical experience and of our own laboratory results, I believe, and for the purposes of this discussion shall assume, that the more nearly nitrogen equilibrium is attained—in other words, the more perfectly nutrition is maintained—the greater are the chances in the patient's favor. Accordingly, I shall place the daily requirement of the average adult typhoid fever patient at more than 40 calories per kilogram, but would emphasize the statement that the optimum amount of food can be determined only by the needs of each patient individually.

Patients of small stature, including children, require more energy per kilogram, but not necessarily more actual energy, than average adults, because of the disproportion of surface area to weight.

The Protein, Fat, and Carbohydrate Ratios.—The next question requiring consideration is the relative proportions of protein, fat, and carbohydrate which should enter into the diet of the typhoid fever patient. So little is known concerning metabolism in typhoid fever that a full discussion of the ratios would of necessity be largely theoretical, and therefore unsuited to a paper of this character (4).

Protein.—The best results in the sparing of body protein obtained by Shaffer and Coleman were with diets containing from 10 to 15 grams of nitrogen, or 62 to 94 grams of protein, a day. Since Grafe essentially confirms these results, this quantity may be accepted provisionally as the optimum protein ration. But it is not improbable that the requirement varies in different stages of the disease, and that

even in cases where nitrogen equilibrium has been maintained during the febrile period, there may be an increased demand for nitrogen during convalescence to repair the damage inflicted upon the tissues by the endotoxin.

Gelatin.—Tissue protein cannot be built up from gelatin; therefore gelatin alone cannot supply the nitrogen needs of the body. But Murlin (5) has shown that two-thirds of the minimum daily protein ration may be replaced by gelatin, provided the carbohydrate supply is sufficient, without detriment to health.

While I have used gelatin only to a limited extent, there is no apparent reason why it may not be permitted as a part of the diet for the sake of variety.

Fat.—We possess very little knowledge of the fat requirement in typhoid fever, But physiological economy would appear to make it expedient in fever, as in health, that a portion of the energy of the food should be supplied in the form of fat. Clinical evidence indicates that the fat needs of the typhoid fever patient vary in different stages of the disease. Many, if not the majority of patients, are able to take more fat in the steep-curve period and in convalescence than in the earlier stages of the disease. Fat has often furnished in these periods one-half of the total energy of the food of the cases in the present series, and the patients have appeared to be the better for it.

Carbohydrate.—Of the three classes of foodstuffs, carbyhydrate plays by far the most important part in preventing the consumption of the body tissues in fever. Grafe's studies on the total metabolism of fasting typhoid fever patients agree with the results of Shaffer and Coleman in establishing this fact. It has been found that the amount of carbohydrate necessary to protect the body protein varies in different subjects and at different stages of the disease, therefore only a general recommendation can be made for clinical purposes; that the greater portion of the energy of the diet should be supplied in the form of carbohydrate, unless there is some definite indication to the contrary. A number of patients in this series have taken from 30 to 60 carbohydrate calories per kilogram per day.

Previous Diets.—While it will not be possible to discuss at length the different diets which have been employed in the treatment of typhoid fever, a brief summary of them may be given. Three types of diet hav been used: (1) The meat-broth-carbohydrate-water diet of Graves, which furnished, at the most liberal estimate, not more than 300 calories a day; (2) the milk diet, from which the patient receives about 1,300 calories a day, assuming that he takes two quarts, and (3) the so-called liberal diets. These diets furnish at most only about 2,000 calories a day. They were constructed upon empirical data, and, as might be expected, are open to criticism. More attention has been devoted to increasing the variety of foods allowed than to the dynamic value of the diet as a whole. Likewise, undue emphasis has been placed upon the necessity of increasing the protein.

The history of the typhoid diets thus indicates a definite though gradual tendency to raise the energy value of the food. The change from Graves' diet to milk added approximately 1,000 calories a day to the patient's dietary; the change from milk to the liberal diets added about 700 more. But according to the results of Shaffer and Coleman, the most generous of these diets falls short of the patient's needs by about 1,500 to 2,000 calories a day.

The Selection of the Diet.—The most important considerations governing the selection of foods for the typhoid fever patient are their digestibility, absence of harmful residue, fuel value, and palatability.

Protein Foods.—The choice of protein foods lies among the meats, eggs, milk, and the proteins contained in the carbohydrate foods.

Meats.—The objections which may be brought against meat and preparations of meat raise the question whether they should be given at all to patients suffering from typhoid fever. Meats differ so much in digestibility, according to variety, "cut," method of cooking, etc., that it is difficult to make a comprehensive statement regarding them. Some meats are relatively digestible in health, others appear to be digested readily even in disease. Probably the majority of physicians believe that patients suffering from febrile diseases are better off without meat. When meats are allowed to fever patients, there is likely to be an excess of protein in the diet, and Ewing and Wolf (6) have shown that a relative excess of protein may cause serious disorders of metabolism in severe cases of typhoid fever, especially if the patients are under nourished. Furthermore, the products of the putrefaction of meat in the intestine may not only give rise to digestive disorders, but may irritate the kidneys during their excretion andproduce albuminuria. Meat extracts, such as beef juice and bouillon, have often been recommended, and sometimes have constituted the sole article of the typhoid diet. While the use of small quantities of these preparations may be advantageous at times to stimulate the appetite, two objections may be raised to them--the small amount of energy they furnish and the high percentage of extractives they contain. Beef juice as ordinarily prepared furnishes 25 calories to each 100 c.c. (3 ounces); boullion furnishes only 8. The extractives in beef juice and in bouillon average about 2 per cent. Beyond stimulating the appetite, they serve no useful purpose, and may prove toxic.

Since meat and meat preparations are not necessary in order to supply the typhoid fever patient with nitrogen, the objections which have been raised to them render it advisable that they be excluded from the diet or at least that they be given with caution.

Eggs.—Egg albumin water has been used extensively in the treatment of typhoid fever. White of egg, shaken up with ice, flavored with lemon or sherry and sugar, and strained, has also been recommended. But the value of whole eggs has apparently been overlooked, though they have been employed extensively in other febrile diseases, as, for example, in septic diseases and tuberculosis. Aufrecht and Simon (7) have shown that lightly boiled and raw eggs have a higher food value as part of a mixed diet than a corresponding amount of meat, and that lightly boiled eggs are slightly more digestible than raw eggs. The digestibility of boiled eggs is somewhat lowered by the addition of butter.

Patients with typhoid fever take readily and digest from four to six eggs a day. Eggs may be used to supply not only nitrogen, but energy. Six eggs, for example, furnish 7 grams of nitrogen and 480 calories.

Fats.—The most suitable forms of fat for the typhoid fever diet are cream, butter, and yolk of egg. The principal objections to fat are that it may cause nausea, vomiting, other digestive disturbances, and diarrhea. I shall not enter here into a discussion of the disputed question whether fat is capable of causing disturbances of metabolism. It will only be necessary to say that there has been no evidence in any of the cases of this series of such disturbances, though many of them took large amounts of fat.

The tolerance for fat in typhoid fever is much greater than has been supposed. I have often given as much as 200 to 250 grams of fat a day without producing disturbances of any kind. Yet fat has shown a tendency to cause nausea and vomiting and diarrhea when the quantity has been carried too high. These symptoms, how-

ever, have always subsided promptly when the fat was diminished or stopped. Patients differ in their behavior toward fat, and the fat tolerance must be determined in each case.

Milk.—Milk has been used probably more than any other food in the treatment of typhoid fever, yet few subjects provoke more discussion in medical assemblages than the value of milk in this disease. Many persons declare they cannot take milk even in health. Many physicians believe that milk always produces digestive disturbances and tympanites in typhoid fever. The majority of typhoid fever patients taking the milk diet grow very tired of it before they are permitted to have other food. This, however, is an argument not so much against milk as against the practice of limiting the patient to one article of food.

Milk is an important, but not essential, constituent of the high calory diet. Because of its importance, the value of milk and the arguments against it must be considered at some length. Van Noorden (8) states that the idea which many people have that they cannot take milk is "purely imagination; * * * there are almost no people who do not bear milk well, or who cannot accustom themselves to its use. Peabody (9), in an article protesting against milk as an exclusive diet in typhoid fever, says: "Milk will always remain the most serviceable general food in disease, and especially in fever, largely because it is swallowed with much less effort than attends the taking of other foods * * * and because it is so commonly well borne." Pavlov (10) states that "milk causes a secretion both from the stomach and pancreas; * * * milk excites not only a really effective, but also a very economic secretion; * * * a much larger fraction of its nitrogen is free for use by the organism than with any other kind of food. How admirably, therefore, the food prepared by nature subserves its purpose when compared with all others."

In rare instances, however, persons exhibit a definite idiosyncrasy against milk. Halberstadt (11) considers the idiosyncrasy to be a symptom of a congenital constitutional anomaly, comparable to anaphylaxis. The idiosyncrasy may be against the albumin, fat, or whey. In some instances the deleterious effects of milk are thought to be due to a change it causes in the flora of the intestine. Definite poisoning occurs in these cases, often with inflammatory changes in the alimentary tract, and they must not be confounded with patients in whom, it is claimed, digestive disturbances and tympanites occur from the use of milk. Tugendreich (12) has described a similar poisoning by buttermilk under the title of "buttermilk fever."

With regard to the statement that milk always produces digestive disturbances and tympanites in typhoid fever, each physician will probably be guided by his own experience. There is no inherent reason why milk should be more indigestible in typhoid than in any other fever. My experience is in favor of milk. For years before the development of the high calory diet I gave milk exclusively to patients suffering from the disease. Since 1907 milk has formed an important part of the diet. Yet it is a familiar fact that there is a limit to the quantity of milk which patients can digest. When this limit is overstepped, the bulk of the stools increases, milk curds appear, and diarrhea and tympanites may occur. But to say that a patient cannot take milk because excessive quantities cause alimentary disturbances, is not a well-considered argument. In addition to the fear of causing such disturbances, excessive quantities should be avoided, for the reason that the total potential energy of the milk is not utilized, as is shown by the waste in the stools.

What quantity of milk is to be considered excessive can be determined only by the ability of the individual patient to digest it. This ability probably varies under different conditions. In my experience the average patient with typhoid fever is able to digest 1½ to 2 quarts of milk a day without difficulty.

The argument that milk favors the growth of the typhoid bacillus in the intestine is largely theoretical, and is based upon the discarded conception that typhoid fever is an intestinal disease.

While milk is not an essential constituent of the high calory diet—no article of food is—it will be found difficult to supply a patient with sufficient energy on a milk-free diet without unduly disturbing the protein, fat, and carbohydrate ratios. But after all has been said, the quantity of milk in the diet must be arranged according to the tolerance of the patient for it. If it is known to produce digestive disturbances, it should be peptonized, or the quantity diminished, or it should be stopped altogether.

Carbohydrates.—The carbohydrate supply may be obtained from the starches or sugars, or both.

Starches.—Only such starchy foods should be selected as are easily digested and contain no cellulose. Foods with a low percentage of water are to be preferred, as otherwise their bulk is objectionable. Crackers and toast are especially valuable. Well-boiled cereals (without cellulose), rice, baked or mashed potato may be given in moderation for sake of variety. The only objection to starchy foods in typhoid is their bulk.

Sugars.—In order to meet the carbohydrate demands of the patient, one or more of the sugars must be employed. For practical reasons the choice lies between cane-sugar, lactose, and glucose. Cane-sugar is so sweet that it soon palls upon the taste when given in quantity. Opinions differ as to whether it ferments readily in the intestine. Jacobi (13) states that it ferments less easily than lactose in the case of children. Adults probably differ in their ability to handle it. I have given a tablespoonful at a time in coffee, lemonade, etc., and should not hesitate to increase this amount cautiously. Glucose, because of its taste, likewise soon palls upon the senses. It has not been given to any of the cases of this series.

An impression prevails that lactose does not ferment readily in the intestine, though I have not been able to find the investigations upon which this opinion rests. The typhoid bacillus is not capable of fermenting lactose. In my experience it is not fermented readily in typhoid fever by other organisms than the typhoid bacillus, nor has it exhibited the laxative properties which have been attributed to it. Lactose is not very sweet and does not pall quickly upon the taste. Halasz (14) found that persons in health are able to take 150 grams of lactose at a time without the production of glycosuria, while persons suffering from dilatation or tumors of the stomach were able to take only 20 grams. Bauer (15) found the tolerance for lactose in cirrhosis of the liver to be 80 to 100 grams. I have never given more than 140 grams at a time. In no case of the series, whose urine was examined repeatedly (the urine of about 25 of the cases was examined daily), did glycosuria occur.

It should be added that Langstein (16) is of the opinion that lactose of all the carbohydrates, is least adapted to the child whose nutrition is disturbed, and that cane-sugar is more valuable. Howland tells me that he has found it difficult to give the high calory diet, as here outlined, to young children. Kerr (17), on the other hand, recommends the diet. As no patient of this series was under twelve years of age, I have formed no opinion.

Fruits.—Lemonade, orange juice and orangeade, and apple-sauce have been given to many patients of this series. There has been no reason to think that these fruits have disagreed with them. Fruit has not been permitted, however, or has been given in small quantity and with caution, when patients were suffering from diarrhea.

The foods which have been mentioned above are the only ones which have been employed to a sufficient extent to justify their recommendation, but the diet will unquestionably admit of further elaboration, especially in the matter of variety.

Proprietary Foods.—A great variety of proprietary foods are manufactured. They may be roughly classified as meat extracts, other protein foods, some of which have been partially predigested, and foods which have been re-enforced by the addition of carbohydrate or fat, or both. They are marketed as liquids, pastes, and powders. The protein foods are made from meat, eggs, milk, blood, plants, malt, and of mixtures of these. The sources of these foods suggest that their native ingredients may be equally valuable.

Concerning meat extracts, Bigelow and Cook (18) state that "meat juice prepared in the home or hospital * * * is far superior as a food to the commercial meat extracts and so-called meat juices." Many of the liquid preparations contain alcohol—some as much as 20 per cent.—which fact must be borne in mind or an excessive quantity of this drug may be administered. The so-called predigested protein foods must likewise be given with caution, since it has been shown that proteoses and peptones may cause diarrhea and other digestive disorders. The work of Ewing and Wolf, as already mentioned, has demonstrated the danger of giving protein to patients who take other foods poorly, and the frequently advertised advice to give such patients proprietary protein foods must be unreservedly condemned.

Lusk (19) states that "the principal value of 'patent' foods lies in their flavor. * * * That beef, milk, cream, butter, and rice are equally suitable for all the purposes of proper living is a fact not sufficiently advertised."

If it should become necessary in any case of typhoid fever to give a patient an artificial food, a preparation in which carbohydrate predominates should be selected, and its fuel value should be clearly understood.

In my experience proprietary foods are unnecessary in the treatment of typhoid fever. I have always found that, if a patient could take food at all, he could take a natural food.

Argument for the High Calory Diet.—The value of the high calory diet must rest ultimately upon the results obtained from its use in a large number of cases. Pending this verdict, estimates of its value may be made from such sources as are available. These sources comprise our knowledge of nutrition, of bacterial infections in general, and the opinions of physicians of experience in the treatment of typhoid fever.

It is axiomatic that life cannot continue unless the body is supplied with energy for its metabolic exchanges. If food is not available in sufficient quantity, the tissues of the body will be drawn upon. Clinical evidence as well as numerous experimental investigations have shown this to be true in fever as well as in health. There is probably no infective disease, except those which affect the alimentary tract locally, which is benefited by partial or complete starvation.

Though typhoid fever often strikes down persons in apparently perfect health, it is generally accepted as true that both the lower animals and men who are undernourished are less able to resist bacterial invasion. Furthermore, healthy laboratory animals furnish a better immune serum than those which are sickly, and the belief prevails that patients suffering from many, if not all, infective diseases are more likely to recover when properly nourished. When in addition we consider that, as Grafe has shown, metabolism in typhoid fever probably follows normal laws, there is no known reason why patients suffering from this disease should be partially starved. On the contrary, and especially in view of the increased metabolism in fevers, all in-

dications point to the desirability, if not necessity, of supplying patients with all the food their require.

Physicians who have raised the nutritive values of their diets are practically unanimous in their opinions as to the beneficial effects of the change. Graves (20) believed that even his meagre diet modified favorably the course of the disease as he knew it. Trousseau (21) shared this belief. In advocating the milk diet, Flint (22) compared the typhoid fever patient to a person in danger of drowning, and said: "As a person in this situation requires only to be buoyed up by some support, so the fever patient in a similar emergency may need only supporting means to live until the disease ends."

Marsden (23) claimed that his patients recovered more rapidly, and that the tendency to asthenic complications was lessened.

Barrs (24) thinks that the sooner a patient returns to a natural diet the sooner will his nutrition be such as to place his intestine under the best possible condition for healing; that free feeding is likely to modify favorably the death rate, to shorten convalescence, and to diminish the risks of complications and sequelae.

Kinnicutt (25) found, in an analysis of 600 cases on liberal diets, that hemorrhage and perforation were less frequent than on a restricted diet, chiefly milk, and that relapses were not increased.

Many other favorable opinions could be cited, but it seems unnecessary. It is a significant fact that the adverse criticisms of the liberal diets have come for the most part from physicians who have not employed them. Few or no physicians who have given these diets an extended trial have returned to the older restricted methods of feeding. If one accepts the foregoing evidence, the high calory diet is deprived of the radicalism which has been attributed to it, the object of the diet being simply to supply patients with all the food they need, instead of supplying them with part of it.

Criticisms of the High Calory Diet.—The only criticisms which have been made of the high calory diet, so far as I am aware, relate to the ability of the patient to digest and absorb the amount of food recommended.

Von Hoesslin (26) proved that digestion in typhoid fever is only slightly below normal, not more than 10 to 15 per cent.

Eugene F. Du Bois has investigated during the last year the food losses in the stools of several cases of this series. The results will be published in full by him later on, but a brief summary of them may be given here.

The protein losses, with patients taking from 65 to 118 grams, were usually under 10 per cent. The loss in a normal man on the same diet, studied as a control, was 7.9 per cent.

The fat loss in the second week of the disease, when the temperature was high, with patients taking from 147 to 200 grams, averaged 8.84 per cent. The loss in the steep-curve period of the third and fourth weeks, with the patients taking from 150 to 258 grams daily, was 4.15 per cent. The fat loss in the control, taking 164 grams daily, was 2 per cent. According to Rubner, a normal man taking 79.9 grams of fat a day in milk lost 7.1 per cent. in the stools, and a normal man taking 214 grams of fat in the form of butter lost 2.7 per cent.

The carbohydrate loss in all periods of the disease, with patients taking from 215 to 567 grams daily, was only 0.1 to 0.3 per cent. The loss in the control while taking 249 grams was 0.1 per cent.

The results obtained by Du Bois demonstrate that the ability of the typhoid fever patient to digest and absorb the high calory diet is remarkable, and they may be interpreted as an additional indication of the patient's need for such

surprisingly large amounts of food. These results should not be interpreted to mean, however, that the typhoid fever patient may be fed indiscriminately with foods which may be taken by the healthy man with impunity, but simply that carefully selected, easily digested foods may be given to typhoid fever patients (in quantities proportioned to the ability of the patients to digest them) without fear of harm.

The further fact should be noted (and this has been observed by all persons who have been brought in contact with the patients), that the stools of patients on the high calory diet presented an unusually normal appearance.

Administration of the Diet.—A false impression exists concerning the high calory diet, namely, that a patient should be given large amounts of food, especially milk sugar, simply because he has typhoid fever, irrespective of the result produced. The physician should endeavor to maintain nutrition in all cases, but I would emphasize the statement that *no patient should be given more food than he is capable of digesting and absorbing.* If the food is vomited or passes through the intestine unabsorbed, the very object of the diet, improvement of the patient's nutrition, is defeated.

In no case can we do more than attempt to give patients the amount of food they require. Failure to accomplish this may depend either upon lack of proper attention to detail on the part of the physician or to lack of co-operation on the part of the nurse, as well as to obstacles which the disease itself interposes. The physician should possess at least a rudimentary knowledge of the calory values of foods. But probably the chief requisite to the successful administration of the diet is intelligent co-operation on the part of the nurse. When a nurse is trained in the use of the diet, general directions regarding the total number of calories and the protein, fat, and carbohydrate ratios will suffice. At her discretion she will increase or diminish the total amount of food, or of particular articles, until further instructions. When a nurse is not trained in the use of the diet, the physician himself must assume immediate control of the feeding.

Cautions in Giving the Diet.—Whenever the administration of the high calory diet is undertaken, careful attention should be paid to the behavior of the stomach, the condition of the abdomen, and the number and character of the stools. If any article of food causes persistent disturbances of digestion, it should be diminished in quantity or stopped. The qualification "persistent" is made because neither occasional vomiting, slight tympanites, or mild diarrhea has been found to be a contraindication to the diet.

One should proceed cautiously in all cases in increasing the amount of food, but not necessarily slowly. If for any reason a patient takes all food poorly, as in cases with alcoholic or other gastritis, the diet should be reduced to the simplest terms, but the patient should be given all the food he can take. For reasons already stated, such patients should not be fed exclusively upon meat broths or other preparations in which protein predominates. Attention should be directed to the fact that it is not uncommon for patients on the high calory diet to have several formed stools a day. This, of course, is not an indication to modify the diet. Many patients who, while taking the high calory diet, have only one stool a day will have from three to six stools a day when put upon the "regular" hospital diet containing meat and vegetables.

Milk sugar may cause nausea and vomiting, especially at first, if the quantity is increased too rapidly. I have found, however, only a limited number of patients whose stomachs could not be educated to tolerate it, even in large quantities. When

milk sugar causes nausea or occasional vomiting, it is necessary to diminish the quantity or to change the method of its administration. If it causes persistent nausea and vomiting, it should be stopped, but may be begun again in a day or two in smaller amounts. The addition of only a teaspoonful to milk or other suitable articles of food is just so much energy gained. A similar course should be pursued if the milk sugar causes tympanites, though at times, when this has been slight, I have diminished but not stopped it.

Cream may cause gastric disturbances or diarrhea. In the former case it should be stopped; in the latter, diminution of the quantity may be sufficient to bring the diarrhea under control. I have not considered it necessary to modify the diet when patients were having only two or three diarrheal stools a day. Not infrequently patients entering the hospital with diarrhea have had this stop under the cautious administration of the diet.

The Amount of Food.—As has already been pointed out, the amount of food which a patient requires must be determined by his individual needs. The calculated requirement serves only as a general indication of them. The condition of the patients of this series, in whom the nitrogen balance was determined, corresponded in general with the amount of nitrogen lost. Patients losing nitrogen lost weight, and the more nearly nitrogen equilibrium could be maintained, the better was the condition of the patient. But since it is manifestly impossible to determine the nitrogen balance, except in a few cases, clinical guides to the patients' needs must be sought. Two such guides have been found useful—the weight of the patient, and the state of his appetite. It is not practicable to weigh patients in private practice, and the physician must depend upon his judgment to tell whether a patient is gaining or losing in weight. A patient who is losing weight is evidently drawing upon his own tissues, and certainly requires more food.

If hunger has any significance, we must believe that it indicates the need of food. I always try to appease the appetite of a patient who is hungry.

In the earlier stages of the disease, when the temperature is continuously high, it is always difficult and sometimes impossible to give patients the amount of food they require, that is, the amount which will prevent loss of weight. In perhaps the majority of severe cases it will not be possible to give more than 3,000 calories, but as the temperature passes into the steep-curve period, the number of calories may gradually be increased. In this period and in convalescence patients take eagerly from 4,000 to 6,000 calories a day (one patient took 7,400 calories). The question of the advisability of permitting patients to have such large amounts of food has been raised by physicians and others who have seen the cases. In every instance these quantities have been reached only in response to requests from the patients for more food. Some patients have complained of hunger while taking 3,500 to 4,000, and even more, calories a day, and I have seen no reason for attempting to curb their appetites. It is not likely that all this food is used for producing energy. Part of it is probably stored as glycogen and fat. As will be pointed out later, gains of a pound a day in weight are not uncommon in the steep-curve period and in convalescence. If only half of such a gain is represented by fat, approximately 2,000 of the calories would be accounted for, leaving not much more of the potential energy of the diet than is generally conceded to be necessary for the daily expenditures of the patient. Not infrequently patients who have lost flesh during the more active stages of the disease will regain their weight before the temperature reaches normal.

The question of what weight we shall permit the patient to reach presents itself during convalescence in nearly all cases. I have no definite opinion to express upon this question, but because of the after-tendency to obesity in many instances, I have

usually followed the practice of changing or limiting the patient's food when the normal weight has been attained. So far as my information goes, the high calory diet does not predispose to obesity after the fever.

Details of Administration.—The successful administration of the high calory diet depends upon unremitting attention to detail. It is a good plan to make a frank statement to the patient regarding the object of the diet whenever, in the opinion of the physician, this can be done. However, it is not always wise to enter too fully into particulars. Very often I tell patients that the more they eat, the sooner they will get well, and the effect of the suggestion upon the quantity of food which they will take is sometimes surprising. I tell every patient who is capable of appreciating the advice, to ask the nurse for more food if he wants it, and I tell the nurse to give the patient all the food he can digest and absorb.

Typhoid fever patients cannot all be fed alike. Their preferences for and idiosyncrasies to foods are not removed by the fever. Yet often the judicious substitution of one article of food or dish for another will increase the fuel value of the diet by several hundred calories. Sometimes patients who complain that the milk is too sweet when it contains one-half to one ounce of milk sugar will take eagerly from two to four ounces of milk sugar in custard, ice cream, or lemonade. As in every other illness, the physician should permit as great variety of foods as is consistent with the patient's wellbeing.

Ordinarily, when a patient first comes under observation he is put upon plain milk for a day or two. The subsequent procedure depends upon the patient's condition; that is, whether he is suffering from a mild or severe attack of the disease. In the former case he may be allowed foods which require mastication; in the latter, the diet should be liquid.

Foods and Their Calory Values (27).—All of the foods and recipes which follow have been given thorough trial, and are recommended with confidence for appropriate cases.

Name.	Amount.	Calories.
Apple sauce	1 ounce	30
Bread	Average slice (33 grams)	80
Butter	1 pat (1-3 ounce)	80
Cereal (cooked)	1 heaping tablespoonful (1½ ounces)	50
Crackers	1 ounce	114
Cream (20 per cent.)	1 ounce	60
Egg	1 (2 ounces)	80
Egg, white	1	30
Egg, yolk	1	50
Lactose	1 tablespoonful (9 grams)	36
Milk (whole)	(1 pint 350) 1 ounce	20
Potato (whole)	1 medium	90
Potato (mashed)	1 tablespoonful	70
Rice (boiled)	1 tablespoonful	60
Sugar, cane	1 lump	16
Sugar, milk (28)	1 tablespoonful (9 grams)	36
Toast	Average slice	80

Rubner's figures for calculating the calory values of the different foodstuffs will be found useful: 1 gram pure protein furnishes 4.1 calories; 1 gram pure carbohydrate furnishes 4.1 calories; 1 gram pure fat furnishes 9.3 calories. Nitrogen multiplied by 6.25 equals protein.

Food Combinations and Recipes.—For the convenience of those desiring to use the high calory diet, the following combinations of foods are given. They are most useful in the early stages of the disease, or in the case of patients who are unable to take solid food.

	Calories.
For 1,000 calories a day:	
Milk, 1 quart (1,000 c.c.)	700
Cream, 1 2-3 ounces (50 c.c)	100
Lactose, 1 2-3 ounces (50 grams)	200
This furnishes 8 feedings, each containing:	
Milk, 4 ounces	80
Cream, 2 drams	15
Lactose, 6 grams	24
For 1,500 calories a day:	
Milk, 1½ quarts (1,500 c.c.)	1,000
Cream, 1 2-3 ounces	100
Lactose, 3 1-3 ounces (100 grams)	400
This furnishes 6 feedings, containing:	
Milk, 8 ounces	160
Cream, 2 drams	15
Lactose, 16 grams	64
For 2,000 calories a day:	
Milk, 1½ quarts	1,000
Cream, 8 ounces (240 c.c.)	500
Lactose, 4 ounces (125 grams)	500
This furnishes 7 feedings, each containing:	
Milk, 7 ounces	140
Cream, 1 ounce	60
Lactose, 18 grams	72
For 2,500 calories a day:	
Milk, 1½ quarts	1,000
Cream, 8 ounces	500
Lactose, 8 ounces (250 grams)	1,000
This furnishes 7 feedings, each containing:	
Milk, 7 ounces	140
Cream, 1 ounce	60
Lactose, 36 grams (29)	144
For 3,000 calories a day:	
Milk, 1½ quarts	1,000
Cream, 1 pint (480 c.c.)	1,000
Lactose, 8 ounces	1,000
This furnishes 8 feedings, each containing:	
Milk, 6 ounces	120
Cream, 2 ounces	120
Lactose, 1 ounce (30 grams)	120
For 3,900 calories a day:	
Milk, 1½ quarts	1,000
Cream, 1 pint	1,000
Lactose, 16 ounces (480 grams)	1,900
This furnishes 8 feedings, each containing:	
Milk, 6 ounces	120
Cream, 2 ounces	120
Lactose, 2 ounces	240

When the above combinatons are employed, it is generally desirable to add eggs to the diet in order to raise the nitrogen to the desired amount. The eggs may be soft-boiled or be shaken up in any of the above feedings unless distasteful to the patient, though the addition of an egg makes the stronger mixtures very rich. I have given some patients who seemed unable to get enough to eat 4 ounces of milk, 4 ounces of cream, 2 ounces of milk sugar, and an egg at a feeding. Such patients, however, are exceptional.

Milk toast, with the additon of butter or cream, is relished by many patients.

The following ménus were arranged by Miss Mary E. Sheehan, head nurse in Ward A1 of Bellevue Hospital, and have been employed, with such modifications as individual patients required, for the last two years. They may be followed in general at any stage of the disease if the patient is capable of taking solid and semisolid food, and if he is hungry, but are most useful in the later stages, and in convalescence. The night feedings are given when the patients' temperatures are taken in the course of the ward routine.

The diet for this day furnishes 3910 calories.

	Hours.	Total.	Calories.
Milk, 6 ounces	9 a. m.; 1, 3, 7	1,260 c.c.	860
Cream, 2 ounces	10 p. m.; 1, 4	420 c.c.	840
Lactose, 10 grams		70 grams	280
			1,980

At 11 a. m.	Calories.	At 5 p. m.	Calories.
Egg, 1	80	Egg, 1	80
Mashed potato, 20 grams	20	Cereal, 3 tablespoonfuls	150
Custard, 4 ounces	250	Cream, 2 ounces	120
Toast (or bread), 1 slice	80	Applesauce, 1 ounce	30
Butter, 20 grams	150	Tea.	
Coffee.		Cream, 3 ounces	180
Cream, 2 ounces	120	Lactose, 20 grams	80
Lactose, 20 grams	80		640
	780		

At 7 a. m.	Calories.
Egg, 1	80
Toast, 1 slice	80
Butter, 20 grams	150
Coffee.	
Cream, 2 ounces	120
Lactose, 20 grams	80
	510

Milk sugar lemonade may be substituted for the milk mixture at three o'clock.

The diet for this day furnishes 5580 calories.

	Hours.	Total.	Calories.
Milk, 5 ounces	9 a. m.; 11, 1, 3, 7	1,200 c.c.	820
Cream, 2 ounces	10 p. m.; 1, 4	720 c.c.	1,440
Lactose, 15 grams		120 grams	480
			2,740

At 11 a. m.	Calories.	At 5 p. m.	Calories.
Eggs, 2	160	Egg, 1	80
Toast, 2 slices	160	Toast, 2 slices	160
Butter, 20 grams	150	Butter, 20 grams	150
Mashed potato, 70 grams	70	Cereal, 6 tablespoonfuls	290
Custard, 8 ounces	500	Cream, 4 ounces	240
	1,040	Applesauce, 1 ounce	30
		Tea	
		Cream, 2 ounces	120
		Lactose, 20 grams	80
			1,150

At 7 a. m.	Calories.
Egg, 1	80
Toast, 2 slices	160
Butter, 20 grams	150
Coffee.	
Cream, 3 ounces	180
Lactose, 20 grams	80
	650

The diet for this day furnishes 5570 calories. The ménu for the hospital "dinner" calls for chicken, and therefore should not be employed until convalescence is well advanced.

	Hours.	Total.	Calories.
Milk, 5 ounces	9 a. m.; 11, 1, 7	1,050 c.c.	700
Cream, 3 ounces	10 p. m.; 1, 4	630 c.c.	1,260
Lactose, 15 grams		105 grams	420
			2,380

At 11 a. m.	Calories.	At 5 p. m.	Calories.
Eggs, 2	160	Toast, 2 slices	160
Mashed potato, 80 grams	80	Cereal, 6 tablespoonfuls	290
Custard, 8 ounces	500	Cream, 2 ounces	120
Creamed chicken, 1 ounce	50	Lactose, 20 grams	80
Toast, 2 slices	160		650
Butter, 20 grams	150		
	1,100		

At 3 p. m.	Calories.
Lemonade (lactose, 120 grams)	480

At 7 p. m.	Calories.
Egg, 1	80
Cereal, 5 tablespoonfuls	250
Cream, 2 ounces	120
Toast, 2 slices	160
Butter, 20 grams	150
Coffee.	
Cream 2 ounces	120
Lactose, 20 grams	80
	960

The following recipes were arranged by Miss Edna Cutler, formerly Dietitian to Bellevue Hospital. They were arranged with the double purpose of adding variety to the patient's dietary, and rendering the milk sugar more palatable, thus making it possible to increase the quantity administered. I have tasted most of these dishes, and have found them savory. Any of them may be given to suitable patients at appropriate times.

Cocoa with milk:	
1 rounding teaspoonful of cocoa	50
2 ounces of milk sugar	240
4 ounces of milk	80
2 ounces of cream	120
	490

Mix the sugar and cocoa; cook in the milk until dissolved. Serve with the cream.

Cocoa:	Calories.
1 heaping teaspoonful of cocoa	50
2 ounces of milk sugar	240
½ cup of water.	
3 ounces of cream	180
	470

Mix the cocoa and sugar, add the water, and boil. Then add the cream, or use less cream and serve with whipped cream.

Coffee:	Calories.
1½ ounces of milk sugar (30)	180
4 to 5 ounces of strong coffee.	
2 ounces of cream	120
	300

Plain junket or rennet custard:	Calories
25 grams (—1 ounce) of milk sugar	100
5 ounces of milk	100
¼ junket tablet.	
1 ounce of cold water.	
Few drops of vanilla.	
	200

See directions for cocoa junket.

Cocoa junket:	Calories
1 teaspoonful of cocoa	50
25 grams of milk sugar	100
5 ounces of milk	100
¼ junket tablet dissolved in 1 ounce of cold water.	
	250

Mix the cocoa and sugar, add the milk, and heat lukewarm, stirring constantly; add the dissolved junket, stir thoroughly, and leave in a cool place to set.

Soft custard:	Calories.
1 cup of milk (8 ounces)	160
1 egg	80
2 ounces of milk sugar	240
Speck of salt.	
2 to 3 drops of vanilla or	
Caramel made of 3 tablespoonfuls of granulated sugar	20 (?)
	500

Beat the egg slightly, add the sugar, salt, and hot milk slowly. Cook in a double boiler, stirring constantly, until it thickens a little (if cooked too long, the custard will curdle, but may become smooth again if set in a dish of cold water and beaten at once). Flavor and cool.

To make caramel: Put the sugar in a pan directly over heat and burn until a very dark brown. Dissolve in hot water or milk.

Baked custard:	Calories.
1 1-3 ounces of milk sugar	160
6 ounces of milk	120
1 egg	80
Nutmeg or vanilla.	
Speck of salt.	
	360

Beat the egg slightly. Warm the sugar and milk, stirring constantly, add to the egg, strain into a custard cup, and flavor. Bake in a pan of water in a moderate oven until a knife when cut into it will come out clean (30 minutes to 1 hour).

Bread pudding:	Calories.
1½ ounces of milk sugar	180
6 ounces of milk	120
1 egg	80
1 slice of bread (⅜ inch thick)	60
½ ounce of butter	120
	560

Spread the bread with butter and cut into squares. Beat the egg slightly; heat the milk and sugar, stirring constantly; mix with the egg and pour over the bread. Grate nutmeg over the top, and bake the same as the custard.

Vanilla ice cream:	Calories.
4 ounces of cream	240
2 ounces of milk	40
2 ounces of milk sugar	240
Speck of salt.	
Few drops of vanilla.	
	520

Mix the cream, milk, and sugar, and heat, stirring constantly, until the sugar is dissolved. Then flavor, cool, and freeze

Lemonade:	Calories.
4 ounces of milk sugar (31)	480
7 ounces of cold water.	
2 tablespoonfuls of lemon juice (or to taste).	

Boil the sugar and water for two minutes, add lemon juice to taste, strain, and cool.

Analysis of Cases.—Conclusions drawn from statistical studies of typhoid fever are apt to be misleading unless the number of cases is large and unless they cover many years. Epidemics differ in severity, and the frequency of complications and relapses varies in different years. Yet the effect of raising the nutritive value of the diet in even a limited number of cases may furnish important evidence of the results to be anticipated in other cases. The fact that metabolism apparently follows normal laws in typhoid fever justifies a comparison of the effects of starvation in this disease with the effects of starvation in health. It is not necessary to starve the race in order tot determine that starvation is detrimental. Analogously, if a few typhoid fever patients are benefited by improving their nutrition, the assumption that the majority will be, at least appears probable.

There are 111 cases in the series.

Character of Illness.—Thirty-seven of the cases were mild, 43 were severe, and 31 were very severe.

Mortality.—Eleven of the 111 cases died, giving a mortality of 10 per cent. The simple statement of the mortality, however, does not convey sufficient information regarding the cases. The mortality rate should be compared with that of cases on other diets in the same epidemics, and the characters of the disease in the cases furnishing the mortality should be considered.

For purposes of comparison, a table of all cases treated in Bellevue and Allied Hospitals during the period covered by this report is given (there were so few cases on the high calory diet in 1907 that they are not included in the computation).

Year.	B. H. and A. H. Total cases.	Total deaths.	Diet cases. Total.	Deaths.
1907	...	...	9	0
1908	315	55	23	1
1909	258	37	39	3
1910	302	45	35	7
	875	137	102	11=10.7 per cent.
	102	11		
	773	126=16.5 per cent.		

As will be seen from the table, 16.5 per cent. of all cases in Bellevue and Allied Hospitals in 1908, 1909, and 1910, exclusive of the cases on high calory diet, died, while 10 per cent. of the cases on this diet died.

It seems fair to assume that the type of infection in the different hospitals of the department was essentially the same in the same seasons.

In order that the character of the disease in the cases which died, and their relation to the diet may be appreciated, it will be necessary to give a brief clinical history of each case.

Case I.—Series No. 11, 1908. E. S. Admitted on the tenth day of the disease; died on the thirteenth day; "walking typhoid;" heart in bad condition on admission; patient pale and dyspneic. Diet started on the eleventh day. On the twelfth day intense air hunger developed; the patient's face was drawn and cyanosed: heart sounds were faint, action irregular.

Case II.—Series No. 47, 1909. W. S. Admitted on the seventh day of the disease with lobar pneumonia as a complication; died on the twentieth day. Has used alcohol to excess; became irrational shortly after admission. Temperature ranged up to 106 degrees F. Diet was taken indifferently. Prognosis very grave from day of admission.

Case III.—Series No. 51, 1909. C. McG. The patient had been on a ten-day spree, lasting until the ninth day of the disease; severe diarrhea during the debauch. Admitted on the fourteenth day with alcoholic gastritis and persistent nausea and vomiting. Food was taken indifferently. Perforation occurred on the twentieth day; operation was performed immediately; death on the twenty-fourth day.

Case IV.—Series No. 74, 1909. A. S. Admitted on the eighth day of the disease; died on the fiftieth day. Temperature range high; dropped to normal on the morning of the thirty-fourth day, but evening temperature was never below 101 degrees F.; rose in a few days to its former high level. Persistent delirium and diarrhea. Developed double otitis media on the twenty-second day. Took food only fairly well.

Case V.—Series No. 77, 1910. J. R. Admitted on the eighteenth day of the disease. Temperature very high; patient delirious. Developed pulmonary edema, and died on the twenty-second day. At autopsy, acute bronchitis, extensive bronchopneumonia, chronic otitis media, and acute parenchymatous nephritis were found as complications.

Case VI.—Series No. 89, 1910. T. K. Very alcoholic on admission; irrational at times. History indefinite, probably in the fourth week of the disease. Persistent nausea and vomiting; unable to take much food. Passed a few small blood clots on the seventh day in hospital; severe hemorrhages on the ninth and tenth days. Complained of pain on right side of abdomen on tenth day; distinct mass palpable below umbilicus. Operation for supposed perforation showed this to be a malformed kidney lying across the brim of the pelvis. The patient had status lymphaticus. At autopsy, acute bronchitis and bronchopneumonia were found as complications.

Case VII.—Series No. 91, 1910. J. S. Admitted on the fifteenth day of the disease. Had status lymphaticus. Patient resisted all treatment, and took very little food. Developed bronchopneumonia about the thirty-seventh day, and died on the thirty-ninth.

Case VIII.—Series No. 92, 1910. T. R. Admitted on the ninth day of the disease; during the day the patient had an intestinal hemorrhage, and pulse became weak. On the following day the patient passed 20 ounces of dark-red blood. Developed diphtheria on the thirteenth day. Died on the twentieth day.

Case IX.—Series No. 92, 1910. J. E. Admitted to surgical ward with abscess of prostate twelve days before the typhoid developed. Temperature fell slowly to normal after operation, but immediately rose again. Transferred to medical ward on the ninth day of the typhoid. Operation wound discharging up to the eighteenth day. Took diet well. Developed lobar pneumonia on the twenty-third day; died on the twenty-sixth.

Case X.—Series No. 94, 1910. A. H. Admitted on the tenth day of the disease. Diet started at once and was taken well. Had severe intestinal hemorrhage on the fourteenth day; blood continued to appear until the twenty-first. On the twenty-second day the temperature dropped to 98 degrees F.; respirations became rapid and shallow; patient died on the twenty-third day. The diet was stopped on the occurrence of the hemorrhages.

Case XI.—Series No. 107, 1910. J. N. Admitted on the eighth day of the disease. Diet started at once; patient was hungry and took food well. The disease ran an unusually long course. Lobar pneumonia developed, and the patient died on the forty-fifth day.

These histories show that Cases IV, X, and XI were the only ones in which the diet had a fair trial. The other cases have not been excluded from the series because similar ones undoubtedly exist among those not on the diet (the details of which have not been accessible), and because of possible bias. If one excludes all deaths except Cases IV, X, and XI, the mortality rate is only 2.7 per cent.

The number of cases in the series and the length of time that the high calory diet has been on trial—four years—apparently justify the assumption that the mortality from typhoid fever may be reduced by maintaining the patient's nutrition at the highest possible level.

Relapses.—Relapses occurred in 23 of the 111 cases, or in 20 per cent. The incidence of relapses by years was as follows:

	Cases.	Relapses.	Per cent.
1907	9	2	22
1908	28	10	39
1909	39	9	23
1910	35	2	5
		Average	20

The percentage of relapses in the first three years is so high that information was sought regarding their occurrence on the First Medical Division (32). The result is as follows:

	Cases.	Relapses.	Per cent.
1907	51	13	25
1908	31	10	32
1909	36	6	16
1910	46	15	32
		Average	26

I do not believe that one is justified in drawing any conclusion from the comparison of these statistics except that the percentage of relapses was

high on both the milk and high calory diets among the cases cited. The comparison may indicate, however, that neither the milk nor the high calory diet has any effect upon the incidence of relapses.

Complications.—Hemorrhage. The occurrence of hemorrhage in the different years was as follows:

	Cases.		Per Cent.
1907	9	1 with hemorrhage	11
1908	28	5 with hemorrhage	17
1909	39	2 with hemorrhage	5
1910	35	4 with hemorrhage	11
		Average	12

Three cases complicated by hemorrhage died from other causes; 1 case died as the result of hemorrhages.

A striking fact has been observed in the cases complicated by hemorrhage. When the patient has been well nourished, the loss of blood has produced little, if any, more effect than the loss of a corresponding amount of blood would produce in a healthy man.

Other complications. Perforation occurred in 1 case, lobar pneumonia in 6 cases, otitis media (probably the lighting up of an old process in all instances) in 9 cases, bronchopneumonia in 4 cases, cholecystitis in 2 cases, pulmonary tuberculosis in 1 case, tuberculous abcess about rectum in 1 case, pleurisy in 2 cases, diphtheria in 2 cases, phlebitis in 3 cases, periosteitis in 1 case, panophthalmitis in 1 case, and abscess about the rectum (non-tuberculous), 1 case.

Weights of Patients.—Weights of the patients were taken usually every second or third day, on a platform constructed for the purpose. The majority of the patients lost some weight during the course of the disease. The losses corresponded in general with the amount of food they were able to take. In the majority of cases the losses were under 10 pounds, and often did not reach 5 pounds. The greatest loss recorded was 20 pounds, between the ninth and twenty-second days of a thirty-one-day case. The attack was mild though long, and the patient took his food well, reaching 3100 calories on the second day after admission, and 4000 later on. Some patients who lost flesh during the early stages of the disease, recovered it in the steep-curve period. Other patients gained 1 to 2 pounds during the febrile period, and several patients gained in weight during relapses. In some the weight remained stationary.

Gains in weight during the first two weeks of convalescence—when under the method of partial starvation patients usually continue to lose—varied from 3 to 10 pounds. In a few instances temporary losses of 1 to 3 pounds occurred in this period. A number of patients gained 1 pound a day; one gained 3 pounds in two days; one gained 5½ pounds in three days; and one gained 9 pounds in five days.

As the gains in weight were retained by the patients even after the food was changed to the "regular" hospital diet, they may be assumed not to have been due simply to water retention.

The Individual Patient.—The claim has been made from time to time that particular diets shorten the course of typhoid fever, but the evidence for such claims will not bear close analysis. No one can predict how long the fever will last in any given case. There is no reason to think that the high calory diet has had any influence upon the duration of the febrile stage of typhoid fever, but there is strong evidence that it modifies the course of the disease favorably. Though perhaps the majority of patients on the diet lose some flesh, the marked emaciation formerly so characteristic of the disease does not occur.

Patients often retain their facial coloring throughout the disease. Mentally they are alert and take an active interest in their environment. Many of them have been permitted to read the daily papers and magazines without injurious effects. The so-called typhoid state has not developed in any patient who was able to take sufficient food, and has disappeared under the influence of the diet when patients have entered the hospital in that condition. I doubt if a physician glancing casually through the wards would be able to pick out the typhoid fever patients.

The patients have been stronger when they were permitted to get out of bed, usually two weeks after the fever subsides, than similar cases were formerly on the exclusive milk diet. They have left the hospital in shorter time and in better condition. While it has been difficult to follow the patients after they left the hospital, the limited information I have been able to obtain leads me to believe that convalescence has been materially shortened.

While I have believed for several years that the principles underlying the employment of the high calory diet in typhoid fever would be found applicable to other acute infectious diseases, the value of the diet in such cases has not yet been tested sufficiently, so far as I am aware, to justify more than tentative recommedations. I have given the diet to patients with lobar pneumonia, but the number of cases has been small. Tympanites is so common in severe cases of pneumonia, and the respiratory distress it causes is so serious, that one should take especial care not to increase it through the careless administration of food. In all cases where it can be done safely during the course of the disease, and generally in convalescence from it, I would suggest that the energy value of the diet be increased by the addition of carbohydrates, though not necessarily milk sugar. The high calory diet has been found to be beneficial in the severer respiratory infections which are commonly called influenzal, and from which convalescence is often so slow. It has been employed with satisfaction in some chronic diseases accompanied by impaired nutrition, and there is reason to believe that it would be useful in severe septic infections and in some forms of pulmonary tuberculosis. But, as has been stated, the diet has not yet been thoroughly tested in any disease except typhoid fever.

Conclusions.—1. None of the older diets for typhoid fever furnishes the patient with sufficient energy for his metabolic exchanges. Therefore a patient taking any of these diets is compelled to live in part upon his own tissues.

2. The amount of food which a patient requires can be determined only by his individual needs. The clinical guides to these needs are the weight of the patient and the state of his appetite. A patient who is losing weight should be given more food if he can digest and absorb it. A patient who is hungry should be given sufficient food to appease his appetite. In the early stage of severe cases it is always difficult to give more than 3,000 calories a day; in the steep-curve period and in convalescence, patients take readily from 4,000 to 6,000 calories a day.

3. If any article of food causes persistent disturbance of digestion, the quantity given should be diminished, or the food should be st pped; otherwise the object of the high calory diet, the maintenance of the patient's nutrition, is defeated. If a patient cannot take all the food he requires, he should be given all he can digest and absorb.

4. Carbohydrates should furnish the greater part of the energy of the diet. The daily protein ration should not be below 62 grams, nor greatly exceed 94 grams. Clinical evidence indicates that a diet rich in fat may be taken by typhoid

fever patients with benefit. Fat has furnished in some cases from one-third to one-half of the total energy of the food.

5. In the cases studied, the high calory diet has apparently modified the course of the disease, shortened convalescence, and reduced the mortality.

Finally, I wish to express my indebtedness and my thanks to Dr. Dana for the privilege of studying cases in his service; to Drs. W. Murray Kerr, T. J. Kearns, W. E. Lowthian, T. R. Pooley, Jr., Harold De Wolf, and William Tomkins, House Physicians in different years, for valued assistance, and to Miss Mary E. Sheehan, Head Nurse in Ward A1, for many practical suggestions and for her untiring aid in carrying out the administration of the diet.

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27. The calory values given in the table are approximate, for the most part, but are sufficiently accurate for practical purposes. The values stated are based upon the tables of Atwater and Bryant, Schall and Heisler, Arnold's Diet Charts, and upon weights taken in the hospital.

28. For practical purposes, the milk sugar may be measured in a medicine glass. Each measured ounce equals 18 grams in weight. If milk sugar is added to water in the proportion of 24 grams to 30 c.c. and the water brought to the boiling point, the milk sugar is completely dissolved. Such a solution, made daily or just before use, will be found convenient in administering the diet.

29. If this and the following combinations are too sweet, a portion of the milk-sugar may be given in some other form.

30. By previously dissolving the milk sugar in water, 72 grams of it may be put into a cup of coffee.

31. Some patients have said the lemonade as prepared was not sweet enough, at which times 1 or 2 tablespoonfuls of cane sugar have been added.

32. I wish to express my thanks to the visiting physicians on this Division for the privilege of using these statistics.

FIVE YEARS EXPERIENCE WITH THE HIGH-CALORY DIET IN TYPHOID*.

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Three years ago I called attention to a new principle in the dietetic treatment of typhoid, namely, the principle of supplying the patient with sufficient food to diminish materially, and in some cases to prevent, loss of nitrogen and weight.

The amount of food recommended exceeded that furnished by any diet hitherto employed in the treatment of the disease by 1,500 and 2,000 and more calories a day. Though the number of cases in which the diet had been used was

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not large, something less than fifty, the results had been so striking that it seemed desirable to advocate the principle publicly.

In the discussion which followed the reading of the paper, criticisms were made of the diet which, had they been justified, would have rendered culpable any further attempt to employ it.

In the three years which have elapsed since the paper was presented, the study of the effects of the diet has been steadily pursued. The purpose of the present review is to report briefly the results which have been accomplished: The number and variety of foods employed have been increased; the absorption of the food has been studied by my assistant, Dr. Eugene F. DuBois; certain phases of the metabolism of patients while on the diet have been investigated with the aid of the "small" Benedict respiration apparatus, and clinical material has been accumulated. I shall consider, also, the most important criticisms which have been made.

The number of foods originally employed was limited. With added experience the number has been increased, until now the diet furnishes considerable variety. The following foods have been given thorough trial and are recommended for appropriate cases.

The calory values given in Table 1 are approximate, for the most part, but are sufficiently accurate for practical purposes. The values stated are based on the tables of Atwater and Bryant, Schall and Heisler, Arnold's diet charts, and on weights taken in the hospital.

Other articles of food will probably be added from time to time as the effects of their administration can be observed. At present, the foods which I believe most likely to prove harmful are meat and its preparations (except small quan-

TABLE 1.—FOODS AND THEIR CALORY VALUE.†

Name.	Amount.	Calories.
Apple sauce	1 ounce	30
Bread	Average slice (33 grams)	80
Butter	1 pat (1-3 ounce)	80
Cereal (cooked)	1 heaping tablespoonful (1½ ounces)	50
Crackers	1 ounce	114
Cream (20 per cent.)	1 ounce	60
Egg	1 (2 ounces)	80
Egg, white	1	30
Egg, yolk	1	50
Lactose‡	1 tablespoonful (9 grams)	36
Milk (whole)	(1 pint 350) 1 ounce	20
Potato (whole)	1 medium	90
Potato (mashed)	1 tablespoonful	70
Rice (boiled)	1 tablespoonful	60
Sugar, cane	1 lump	16
Sugar, milk (28)	1 tablespoonful	36
Toast	Average slice	80

tities of meat broth, given for the purpose of stimulating the appetite and for sake of variety), vegetable foods containing much cellulose, and fruits containing much cellulose and small seeds, such as berries.

The three most important objections which have been brought against the

† Published in Am. Jour. Med. Sc., January, 1912.

‡ For practical purposes, the milk-sugar may be measured in a medicine glass. Each measured ounce weighs 18 gm. If milk-sugar is added to water in the proportion of 24 grams to 30 c.c. and the water brought to the boiling point, the milk-sugar is completely dissolved. Such a solution, made daily or just before use, will be found convenient in administering the diet.

diet are: (1) that patients cannot digest and absorb the amount of food recommended; (2) that the amount of fat would inevitably cause alimentary disorders and acidosis, and (3) that, granting its absorption, patients do not require the amount of food which is advocated.

These objections arose in our own minds during the course of our investigations; one of them (the development of acidosis) had been answered by the careful studies of Dr. Shaffer; all of them appeared, on the basis of clinical observation, to be unfounded.

I. Digestion and Absorption in Typhoid Patients.

The objection that patients cannot digest and absorb the amount of food recommended is based on the prevalent notion that typhoid causes serious impairment of the digestive powers. This belief appears to be without foundation. While there can be no doubt that food may cause disorders of digestion in typhoid, their occurrence depends, in my experience, not so much on the quantity as on the method of giving it. Unsuitable foods will, of course, prove harmful.

Through the investigations of Pavlov we know that the digestive glands adapt their secretions to the kinds of foods which they are required to digest, and that changes in diet are followed by alterations in the characters of the digestive juices. These alterations take place gradually and sudden changes in diet, especially from a sparse to a rich diet, are likely to cause digestive disorders. Furthermore, individual peculiarities of digestion must be taken into consideration. These can be discovered only by testing the capacity of each patient for the foods allowed. But if proper attention is paid to these details, I believe it will be found that the great majority of patients may be given the amount of food they require not only without causing disorders of any kind, but with the disappearance of disturbances which previously existed. The studies of Kendall (1), on the influence of diet on the intestinal flora, apparently furnish the explanation of the beneficial action of large amounts of carbohydrate on the condition of the intestine in typhoid. According to Kendall the presence of available carbohydrate protects protein from the putrefactive activities of intestinal bacteria and prevents the disturbances which would result therefrom.

Intimately related to the ability of the patient to digest large quantities of food is his capacity to absorb them. Von Hoesslin investigated this problem thirty years ago and found that the digestive powers of the typhoid patient were only from 10 to 15 per cent. below normal but the differences in the characters of the diets employed by von Hoesslin and by ourselves made it desirable that the subject be reinvestigated. As already stated, this has been done by DuBois, who will shortly publish his results. Briefly, they prove that the capacity of the typhoid patient to absorb large amounts of food is remarkable. The absorption of carbohydrate was practically complete, less than 0.5 per cent. being lost. The average loss of protein was 7.1 per cent. The average loss of fat in the active period of the disease, when the patients were taking from 147 to 200 gm. was 7.2 per cent.; in the steep-curve period and in convalescence, when the patients were taking from 150 to 258 gm., it was 4.5 per cent. The normal loss for similar diets is about 3 per cent., but, according to Rubner, the loss of fat may reach 7.1 per cent. in health.

II. Amount of Fat.

The objection that the amount of fat recommended would inevitably cause alimentary disorders and acidosis has likewise been proved without foundation. The majority of patients treated have taken from 100 to 250 and more gm. of fat a day without disturbances of any kind. It is well known that fat inhibits the

secretion of hydrochloric acid and delays the passage of chyme from the stomach, but neither of these actions has caused inconvenience to the patients. Moderate diarrhea has occurred in a few cases, but has ceased on withdrawing the cream. Duodenal regurgitation has not been observed.

Instead of interfering with digestion, there is reason to believe that fat aids the digestion of carbohydrate. Mixtures of fat and protein are known to be difficult to digest, but, according to Pavlov, the addition of butter (fat) to bread (carbohydrate) facilitates its digestion. The fat delays the passage of the carbohydrate from the stomach while the pancreas is elaborating the ferments for the digestion of both.

I cannot discuss at this time the general question whether large quantities of fat are capable of causing acidosis. The point at issue is whether fat, in the amounts given, causes acidosis in typhoid fever. By way of answer, it may be stated that no patient has presented any clinical evidence of the condition, the acetone bodies have not appeared in the urine and the ammonia nitrogen of the urine in some thirty cases has never exceeded 2 gm. and has usually been below 1 gm.

III. Requirement of Food.

It will be necessary to consider from the clinical as well as from the metabolic standpoint the objection that, granting its absorption, patients do not require the amount of food which is advocated. The effect of the diet on the patient has dominated the investigation from its inception. In general, the more food a patient takes, the better his physical condition. Patients lose weight when an apparent excess of food is not given. The largest amounts which have been administered have been reached in the attempt to satisfy the patients' hunger. Though this is clinical evidence, it possesses considerable value, the ultimate test of any method of treatment being the effect it produces on patients.

The study of the protein metabolism in typhoid has demonstrated that large amounts of food are required to keep a patient in, or nearly in, nitrogen balance. The maintenance of nitrogen equilibrium in fever probably signifies the optimum state of nutrition.

The total metabolism in typhoid has been studied previously chiefly on patients in the fasting state. Last fall, with Dr. DuBois, I undertook to investigate, with the aid of the "small" Benedict respiration apparatus, the total metabolism of patients on a full diet. Owing to the vastness of the field and the technical difficulties to be overcome, the investigation was confined, temporarily, to the study of the general effects of the diet on metabolism. Interesting results, however, were obtained. The greatest amount of heat produced by any patient was 48 calories per kilogram per day. The majority of patients produced around 35 calories per kilogram. Since the patients were at absolute rest during the period of observation (fifteen minutes), at least 10 per cent. must be added to cover the muscular work incident to moving about the bed. Only one of Grafe's fasting patients produced as much as 40 calories per kilogram; the smallest amount of heat produced by any of his patients during the febrile period was 28.5 calories per kilogram (2). Rolly's figures are essentially similar.

Calculated on the basis of these figures, the high-calory diet furnishes from 1,000 to 2,000 more calories than are expended by the patient in twenty-four hours. So far, it has been found impossible to explain this discrepancy in the early stages of the disease, yet I am convinced from the clinical evidence that patients require the excess—they lose both nitrogen and weight if they do not

receive it. In the later stages of the disease, the excess is utilized for the storing of fat, as is shown by the height of the respiratory quotient. In one instance

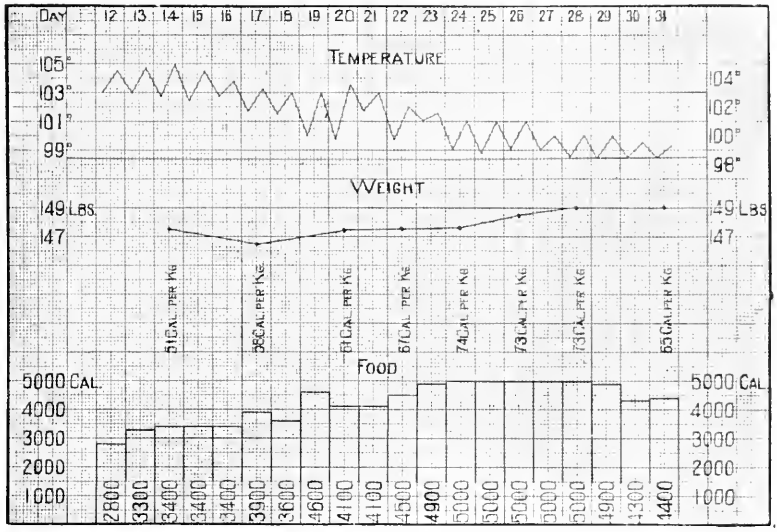

Chart showing temperature, weight and food-curves in a severe case of typhoid.

the actual transformation of carbohydrate into fat was observed while the patient had a temperature of 102 F.

In Bellevue Hospital to Jan. 1, 1912, 138 (3) cases were treated, of which forty-four were mild, fifty-seven severe and thirty-seven very severe. Twelve patients died, giving a mortality rate for the series of 8.69 per cent.

The comparative statistics of the cases of this series and of the department of Bellevue and Allied Hospitals permit the following conclusions:

Table 2.—Comparative Statistics of Typhoid Cases With Regard to High-Calory Diet.

Year.	B. H. & A. H. Total Cases.	Total Deaths.	Diet Cases Total.	Diet Cases Deaths.
1907	...	...	9	0
1908	315	55	28	1
1909	258	37	39	3
1910	302	45	35	7
1911	229	32	27	1
	1,104	169	129	12 =9.3%
	129	12		
	975	157=16%		

Both mortality rates are abnormally high. As probably occurs in other large hospitals, not only the worst types of disease are admitted to the hospitals of the department, but many patients are brought in, apparently only to die, when treatment at home has proved unsatisfactory. All of these cases are included in the figures from which the conclusions are drawn.

Relapses occurred in 20 per cent. of the cases of the series.

Hemorrhages occurred in 13 per cent. of the cases.

Perforation occurred in one case.

The influence of the diet on the weight of patients is shown in the figure.

Reports from Convalescents: During the past year, patients have been re-

quested to report at the hospital at intervals after their discharge. A number of them complied with the request and the records of their condition justify the statement that convalescence is materially shortened.

Since the foregoing communication is in part a review of work already published and in part a report of progress on investigations which are under way, I shall not attempt to draw conclusions from the new matter which it contains.

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WEIGHT CURVES IN TYPHOID FEVER (1).

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While there can be little doubt that under the ancient doctrine of "stuff a cold and starve a fever," loss of weight in the continued fevers was even greater than it is today, I shall confine the present discussion to the period during which typhoid fever has been recognized as a distinct disease—that is since 1837, when Gerhard and Pennock (2) finally established its differentiation from typhus fever. From that time to the present emaciation has been considered a characteristic symptom of the disease. It occurs in greater or less degree in all or nearly all cases. It is prominently mentioned in all complete descriptions of the disease, and has been regarded as a deplorable but necessary consequence of the infection. According to Loomis (3) "Emaciation is perhaps more marked and rapid in this than in any other form of fever. It commences early and is progressive. By the time a patient has reached the fourth week of a typhoid of even moderate severity he is usually in a condition of extreme emaciation." On the other hand, while admitting the occurrence of extreme grades of emaciation, Curschmann thinks that the losses in typhoid fever are not so great as would be anticipated, if one considers the duration of the disease and the extent of the losses in other infections.

Comparatively early in the history of typhoid fever exact observations were made upon the extent of the loss in weight. Yet in view of the fact that the majority of authors do not record the amount of food taken, it is difficult to compare their results. In a few instances, however, they have either plotted out the weight curves or have furnished data from which such curves could be constructed.

The following general summary represents, I believe, the most important results of all previous investigations:

Loss of weight occurs in practically all cases of typhoid fever but varies greatly in extent.

The severer the infection and the longer the duration of the disease, the greater the total loss.

The greatest loss observed by Curschmann (4) at the beginning of convalescence was 41 per cent. of the body weight. Scharlau (5) had previously recorded a loss of 30 per cent. Losses of 19 per cent. in severe, and 10 per cent. in mild, cases are common (Curschmann). On the other hand, the losses may be relatively insignicant, 1.5 per cent. to 3 per cent. in severe and 1 per cent. to 1.1 per cent. in mild cases (Curschmann).

Extracted from The American Journal of the Medical Sciences, November, 1912, No. 5, vol. cxliv, p. 659.

Daily losses of 300 to 500 grams are common; they may reach 1,500 grams (Botkin) (6); and in a case complicated by parotiditis the loss amounted to 2,500 grams in one day (Lorain) (7).

Lorain states that the maximum loss is reached at the end of the second or beginning of the third week, while according to Curschmann it is reached more frequently in the third than in the second week.

Kohlschütter (8) found that, after the more active period of the disease is over, the losses diminish with each week up to complete defervescence. Puritz (9) observed a corresponding diminution in the elimination of nitrogen.

Complications increase the loss of weight (Lorain, Cohin) (10).

While loss of weight usually ends with the return of the temperature to normal, it may extend far into convalescence (Leyden) (11), according to Curschmann to the third week. Curschmann has observed a loss of four pounds in the first week of convalescence.

All patients gain weight in convalescence, some rapidly, apparently without much, if any, increase in the quantity of food.

Contrary to the conclusions of the majority of observers, Garnier and Sabaréanu (12) found in their cases that the weight remained stationary, or increased a little, during the active febrile period; it diminished rapidly at defervescence; and in the afebrile period, remained unchanged until the patient began to take more food, when it increased. They attribute the course of the curve to the retention of water, and its sudden elimination.

Figs. 1 and 2 are taken from Puritz and Lorain. They illustrate the main features of the summary. Puritz's patient received a relatively full diet, taking an average of 160 grams of protein, 60 to 90 grams of fat, and 300 grams of carbohydrate a day. Lorain's patient received only a few spoonsful of milk.

The loss of weight in typhoid fever has been attributed to three factors: (1) Partial starvation; (2) the febrile temperature; and (3) the toxic destruction of protein.

1. Partial Starvation.—Apparently at all periods in the history of typhoid fever it has been recognized that patients suffering from the disease were undernourished. Bretonneau (13) characterized their condition by the term autophagia. Graves

Lorain's Case.

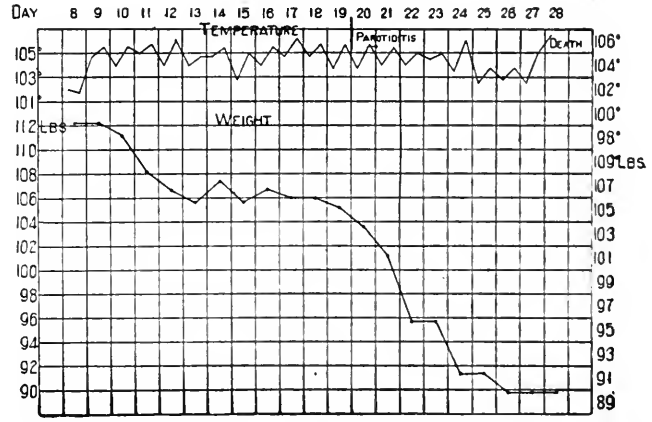

Fig. 1.—Showing influence of complication on weight. Patient received only a few spoonsful of milk a day.

(14), Trousseau (15), Chudnowsky (16), Flint (17), Barrs (18) and Shattuck (19) insisted, in their respective periods, that typhoid fever patients were undernourished, and introduced diets of greater nutritive value.

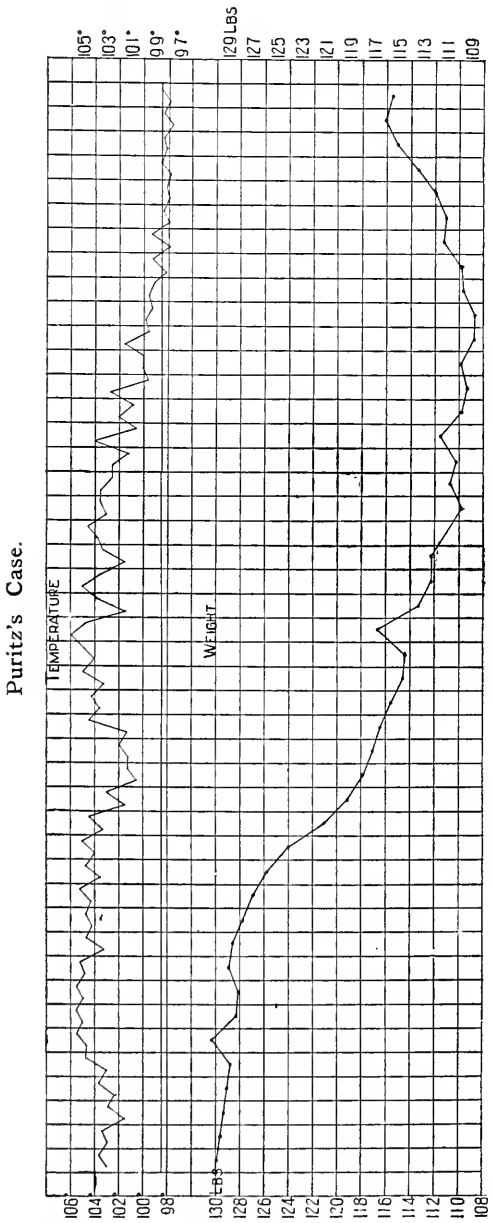

Fig. 2.—Patient received an average of 2000 calories a day.

Proof of the influence of partial starvation upon the loss of weight occurring during the disease is furnished by the observations of Puritz, von Leyden and Klemperer (20), and others who succeeded in diminishing the loss by increasing the

intake of food. Recently it has been found possible to maintain many patients in weight equilibrium by administering sufficient food.

2. The Febrile Temperature.—The effects of an artificially raised temperature (through hot baths, heated air, or injury to the heat centres) upon protein metabolism are comparatively well known. It is probable, but has not been proved, that fat and carbohydrate metabolism are similarly affected. Temperatures under 39 degrees C. cause only slight increase in protein destruction; temperatures over 39 degrees C. likewise have but little effect unless they are prolonged for more than three hours; artificial heating for twelve hours has caused an increase in protein destruction amounting to 37 per cent. (F. Volt) (21).

These results apparently confirm the clinical observation that intermittent temperatures are not, as a rule, attended by such marked loss of weight as those which are continuous. They also suggest that the diminished loss in the later stages of typhoid fever may be due in part to the change in character of the temperature curve.

3. Toxic Destruction of Protein—The loss of nitrogen in the infective fevers is greater than occurs in a healthy man who is starved, or, according to the common belief, than can be accounted for by the pyrexia itself. Upon this basis it has been assumed that the toxins of the infecting micro-organism exert directly a destructive influence upon the cells of the body.

Whether the theory, in its generally accepted form, is correct or not does not concern us at this time. It has been proved possible to bring a patient suffering from typhoid fever into nitrogen and weight equilibrium by the exhibition of a diet containing a large amount of carbohydrate, and a relatively small amount of protein.

Water-Retention Theory.—The discussion of weight curves in typhoid fever would not be complete without reference to the theory of water retention. This theory must be considered from two standpoints: (1) The influence of the disease itself, and (2) the influence of a diet containing a large amount of carbohydrate.

1. The Influence of the Disease.—The water balance has not been thoroughly investigated for any fever, and conflicting opinions are held concerning it in typhoid fever.

Leyden was one of the first to refer to the theory of water retention. The sudden losses of weight which have been observed with remissions of temperature, especially toward the end of the febrile period, probably indicate that water is retained in some cases. Yet Krauss (22) speaks of the "alleged retention of water in fever" as pure "conjecture." Stähelin's (23) dog, infected with surra, eliminated more water than he ingested. Schwenkenbecher and Inagaki (24) conclude from their experiments that water is not retained in the body in fever.

On the other hand, Garnier and Sabaréanu (25) believe that retention of water occurs during the febrile stage of typhoid fever and that this is responsible for the supposed maintenance of weight during this period.

In view of the conflicting opinions and the absence of conclusive studies of the water balance, the question of water retention in fever due to the disease itself must be left open for the present.

2. The Influence of a Diet Rich in Carbohydrate.—The increasing use of diets rich in carbohydrate in typhoid fever renders it important to determine whether such a diet is likely to cause retention of water in the body. As is well known, a poorly balanced diet may cause variations in the excretion of water. For example, in an experiment conducted by Benedict and Milner (26), it was found that water

was retained on the first of three days when the subject was confined to a diet consisting of 965 to 969 grams of carbohydrate; on a diet of 745 to 750 grams of fat, water was lost during the whole period. It is generally agreed, however, that a well-proportioned diet does not affect the water balance.

While the proportion of the foodstuffs in the diet which we employ varies necessarily with different patients, and in some instances has been subject to sudden experimental changes, there has been no constant relation between variation in weight and the quantity of urine. Though water may have been retained by some patients, and have caused an increase in weight, there has been no clinical reason to think that such was the case. There has been no visible edema, and patients have not lost weight suddenly, following diuresis, when the amount of carbohydrate in the diet was diminished during convalescence.

In addition to the above considerations, we now possess evidence that at least a part of the gain in weight in the late stage of the febrile period and in convalescence is due to the conversion of dextrose into fat. In one instance, 38 per cent. of the carbohydrate metabolized during the period of observation (fifteen minutes) was so transformed (27).

The distribution of the losses among the various tissues of the body requires brief consideration.

It appears probable that all of the tissues share in the losses, though to what relative extents can only be surmised at present. The increased elimination of nitrogen in patients who are undernourished, forms the basis for the assumption that the muscles suffer marked loss. Loss of fat, as well as of muscle, is evident clinically in severe cases.

With the aid of a respiration chamber, Grafe (28) studied the respiratory quotients of typhoid fever patients in the fasting state, and reached the conclusion that the metabolic processes follow normal laws, though with increased intensity. Rolly's (29) studies on the respiratory quotients of patients from four to six hours after food, and our own studies in Bellevue Hospital upon patients taking a full diet, lead to the same conclusion. If these results are ultimately confirmed, the consumption of the body tissues in typhoid fever, when the patients are undernourished, proceeds in the same manner as in healthy persons who are partially or completely starved. The stores of glycogen are depleted first—probably within a few hours when the fever is high and, at most, within a few days—after which fat and protein constitute the sole sources of the body's energy. The duration of life in a healthy animal which is starved is measured by the amount of fat which the body contained at the start (Lusk, 30). The often noted fact that obese patients do not bear typhoid fever well does not come into consideration here, since the causes of this fact can only be conjectured. Another analogy of the processes of metabolism in typhoid fever to those in starvation must be noted; in partial or complete starvation the body begins, after a time, to economize its available energy by reducing its total metabolism. A similar economy of energy has frequently been observed in typhoid fever. Moos (31) found as long ago as 1855 that the nitrogen losses diminished in the later weeks of the disease. Kohlschütter (32) emphasized his observations that the loss of weight diminishes with each week after the more active period of the disease is past. Cohin (33) called attention to the same fact. This is the probable explanation of the continuance of life in patients who take a wholly inadequate amount of food and yet live from week to week with high fever.

Influence of the High-Calory Diet Upon Weight.—My own observations upon the weight curve in typhoid fever were begun in 1908. A satisfactory balance could

not be obtained upon the market so I had the hospital carpenter construct a wooden table which would rest upon the platform of the ordinary hospital scales. When in position, the table is the same height as the bed, and the weight of a patient may be taken without more disturbance than shifting his position in bed —with much less exertion, in fact, than is required to administer a tub bath.

Weights have been taken of practically all patients, usually every second or third day. The procedure was adopted originally for the purpose of having additional clinical control upon the effects of the diet. Slight sources of error enter into the observations, which are clearly recognized, but which so far it has been impossible to eliminate (34). While such errors may, and probably do, affect the relation of one day's weight to another, apparently they do not affect the general trend of the curve. The errors referred to are:

Febrile Period and Convalescence.

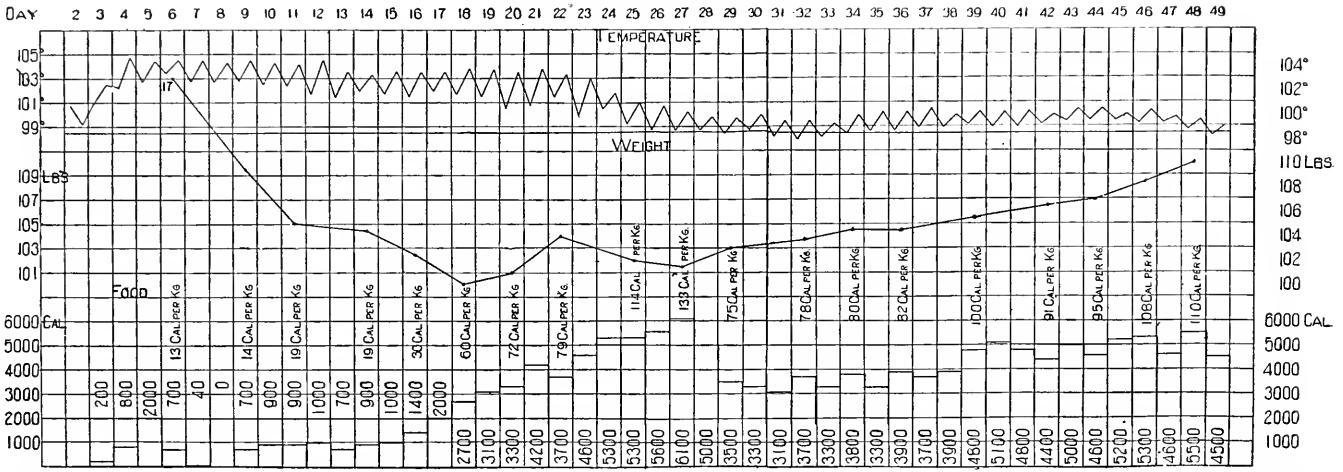

Fig. 3.—One of the earlier Bellevue Hospital cases, showing rapid loss of weight during the febrile period, when the amount of food was insufficient, and the gain in weight in convalescence.

Patients have not always been weighed at the same hour of the day, or with regard to whether they have recently been fed or have urinated. The morning defecations do not come into consideration as the weights have never been taken

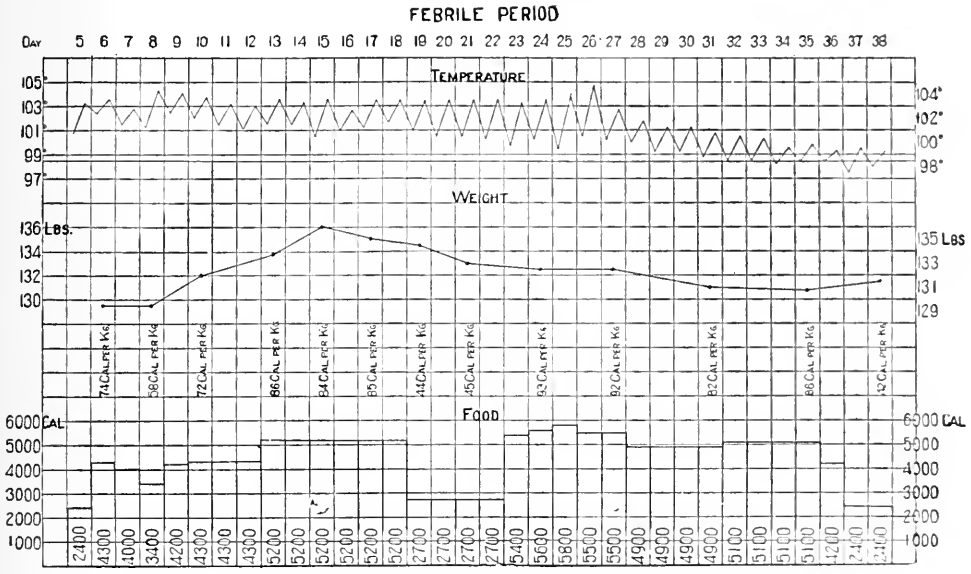

Fig. 4.—Showing that weight equilibrium may be maintained by sufficient food

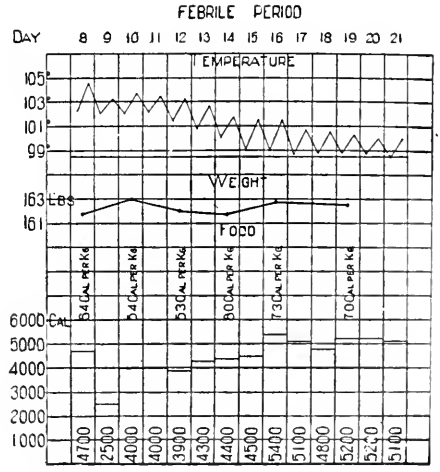

Fig. 5.—Showing the maintenance of weight equilibrium.

before the administration of the daily enema. The clothing of the patients, however, has always been carefully considered. In future observations, it is hoped that these sources of error may be eliminated.

The figures which follow, with the exception of Fig. 3, illustrate the influence of a full diet upon the weights of patients during the febrile period, convalescence and relapses. The curves are typical of the effects of the diet. Fig. 3 is the curve

of one of the earlier patients of the series, to whom it was impossible, with our then limited experience, to give the amount of food required until toward the end of the febrile stage. The general course of the curve is similar to those obtained by the earlier investigators. There is no evidence in the curve of water retention either during the febrile period or in convalescnce.

The gains in weight during relapses and in convalescence are less noteworthy than the maintenance of weight during the active stages of the fever.

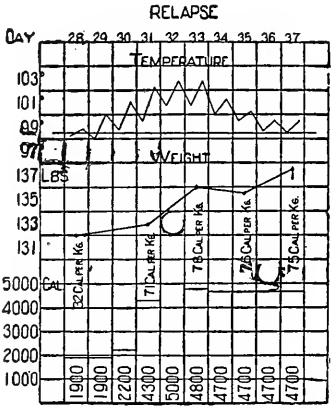

Fig. 6.—Showing an increase in weight during a relapse.

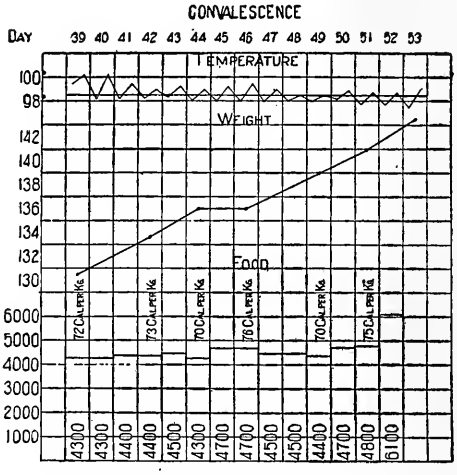

Fig. 7.—Showing rapid gain in weight during convalescence.

The ability of the body to repair its losses after wasting diseases is nowhere better shown than in convalescence from typhoid fever. This fact has been known for many years. Botkin stated that such patients gained weight rapidly at first, more slowly later. According to Liebermeister (35), patients may gain as much as five to seven pounds a week, according to Schottmüller (36), as much as eleven pounds. In Puritz's cases, the weight remained stationary for the first two or three days after the temperature reached normal and then began to increase. Fr. Müller (37) found that patients convalescing from typhoid fever are able to retain nitrogen on a diet which with its low fuel value is insufficient to maintain nitrogen equilibrium in health. He reports a case which retained from the third to the tenth day of convalesence a daily average of 1.3 grams of nitrogen on a diet furnishing only 1,165 calories and 9.9 grams of food nitrogen.

As is illustrated in Fig. 3, patients who have lost much weight during the febrile stage may repair the damage before they are able to get out of bed.

In one of Puritz's cases the weight remained stationary during a relapse of sixteen days' duration.

Conclusion.—It is possible to maintain patients suffering from typhoid fever in weight equilibrium throughout the entire course of the disease by giving them sufficient food.

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THE ABSORPTION OF FOOD IN TYPHOID FEVER*.

EUGENE F. DUBOIS, M.D.

Since von Hösslin and the Russian investigators made their studies on the assimilation of foods in fever twenty to thirty years ago, very little work has been done on the subject. In the meantime, the methods of analysis have been improved and the diet in typhoid has in some clinics been increased so much that patients in the height of their fever are given more food than was formerly given in the second week of convalescence. The question naturally arises as to whether the patients are absorbing the food or are passing it undigested through the intestines.

At the suggestion of Dr. Warren Coleman who has for five years been using a very liberal diet in his service at Bellevue Hospital, I undertook a study of the question in his wards. Six patients with typhoid were studied over periods lasting from five to twenty-one days. They were fed the so-called "high calory diet," which consists of about 1,000 c.c. milk, 300-400 c.c. 20 per cent. cream, 100-200 gm. lactose, two or three eggs, a couple of slices of toast and some butter. This furnishes between two and three thousand calories and one or two thousand calories more can be added in the form of larger amounts of the above or in the form of boiled rice, oatmeal, mashed potato, cream of wheat, apple sauce, custard or ice cream. This diet has been fully described by Shaffer (1) and Coleman (2) and is the only recorded diet which has succeeded in maintaining the patients in nitrogen and weight equilibrium. Clinical experience has shown

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*From the department of applied pharmacology and the second medical division of Bellevue Hospital, New York. All analyses were made in the laboratory of the department of physiology. I am indebted to Professor Graham Lusk for the privilege of working in his laboratory and for many valuable suggestions during the course of the work; also to Dr. Coleman and Dr. Dana for permission to use patients, in their wards, and to Mr. Rudolph H. Harries and Mr. John M. Janson for their assistance in making many of the analyses.

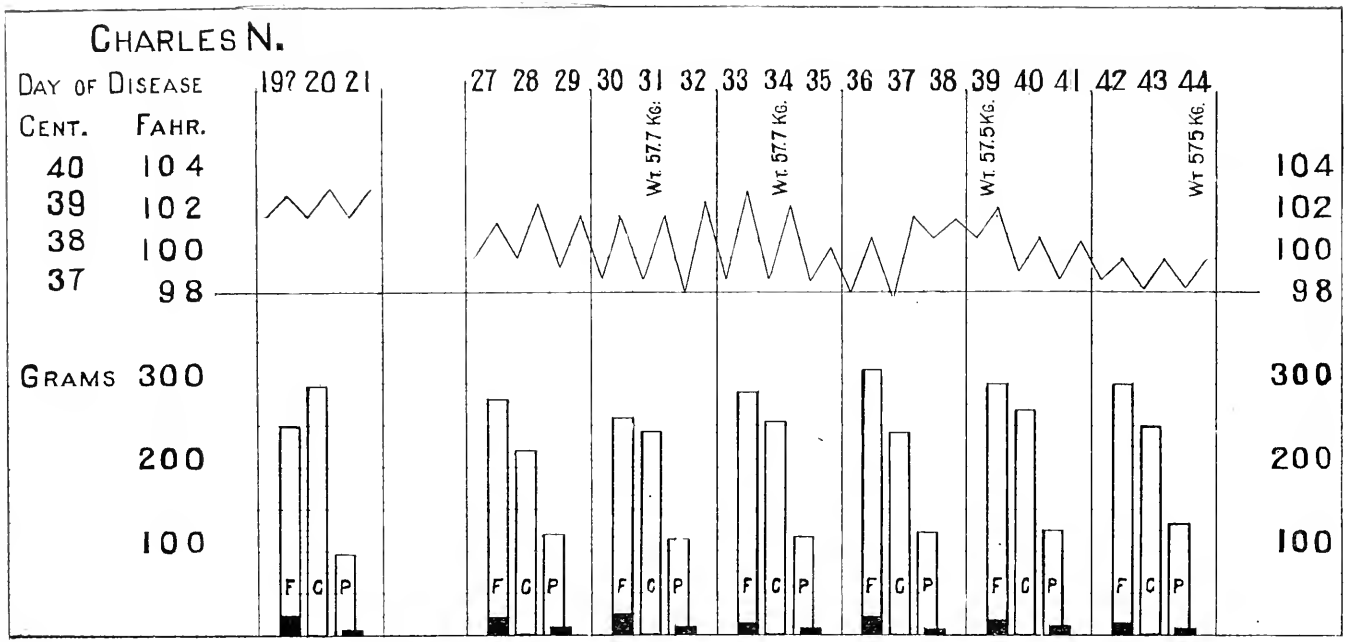

Chart 1.—Temperature curves in Case 1, Charles N The column shows the average daily weight of fat, carbohydrates and protein in food; the solid bases show amounts unabsorbed.

that patients do well on this diet and it was no surprise to find that they could absorb the enormous amounts of food almost as well as normal individuals.

Normal Absorption.

In the case of normal individuals who are given an easily-digested diet, the food is practically all absorbed and the feces consist almost entirely of bacteria and the secretions of the intestinal tracts (3, 4). The feces of a starving person can contain from 0.1 to 0.3 gm. of nitrogen a day, and of a person on a nitrogen free diet as much as 0.5 to 0.87 gm. of nitrogen. Reducing bodies and ether-soluble substances are also present in the stools when none is given in the food. In addition to these secretions of the intestinal tract, however, considerable amounts of food residue do appear in normal persons who are given coarse or poorly-cooked food, or food in unusually large quantities. As a rule, sugars are completely absorbed, and well-cooked starches almost completely. Emulsified fats

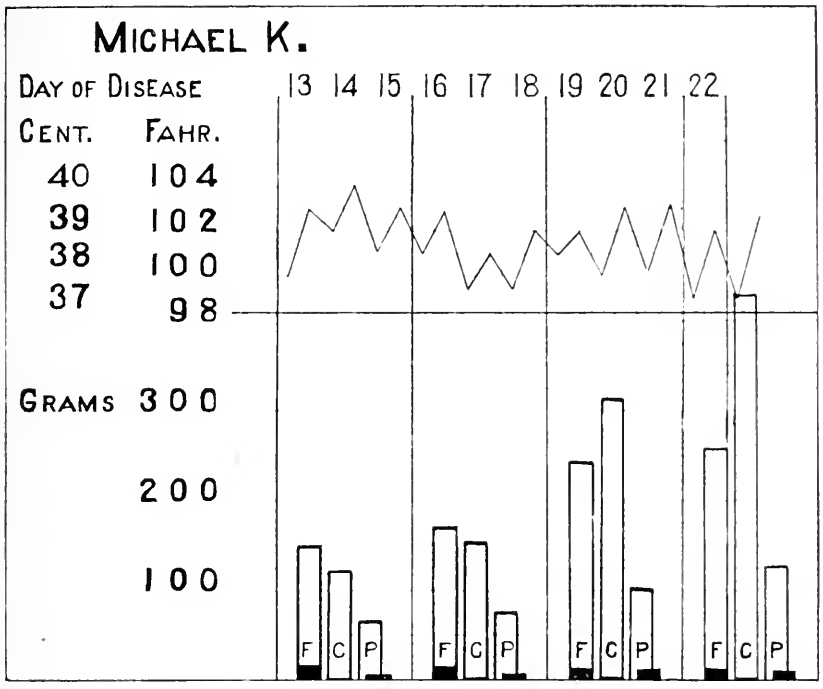

Chart 2.—Temperature curves, etc., in Case 2, Michael K.

of low-melting point are better absorbed than fats of high-melting point. When fats are given in amounts greater than 350 gm. the intestine does not absorb well. (It may be noted that one of the typhoid patients in this series was given 327 gm. of fat on one day.) In considering the absorption of any particular food, such as fat for instance, one can get a false idea if one considers the percentage loss alone without considering both the grams of fat in the food and in the feces. This is shown in a series of experiments by von Noorden (5) on the same individual.

4.2 gm. fat in food—57.1 per cent. loss in feces.
42.2 gm. fat in food—10.9 per cent. loss in feces.
80.2 gm. fat in food—6.36 per cent. loss in feces.

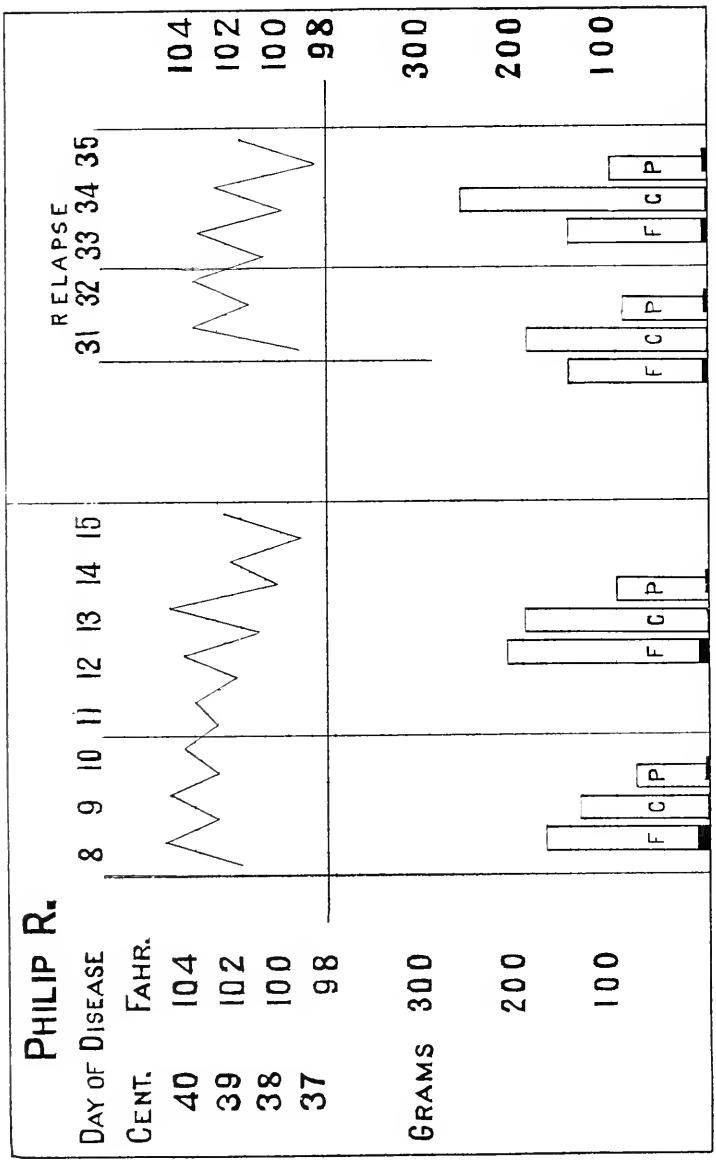

Chart 3.—Temperature Curves, etc., in Case 3, Philip R.

It is hard to state the normal percentage of loss of the various foodstuffs. Rubner (6) gives the accompanying table (Table 1) of the averages of many of his experiments.

TABLE 1.—FOOD ABSORPTION; NORMAL INDIVIDUALS.

Food	Percentage Loss		
	Fat	Carbohydr'te	Protein
Roast beef	....	...	2.6
Hard boiled eggs	4.4	...	2.6
Milk	5.2	0	7.1
Fine white bread	....	1.1	21.8
Rice	....	0.9	20.4
Potato	....	0.7	19.5
Mixed diet with			
Bacon (99 gm. fat)	17.4	1.6	12.1
Bacon (195 gm. fat)	7.8	6.2	14.0
Butter (214 gm. fat)	2.7	6.2	11.3
Butter and bacon (350 gm. fat)	12.7	6.8	9.2

In certain pathological conditions the absorption of food can be greatly interfered with. Obstruction to the flow of pancreatic juice or bile can diminish the absorption of fats greatly. In certain cases of intestinal indigestion carbohydrates are poorly absorbed. Tuberculosis or cancer of the intestine or any very severe diarrhea can increase greatly the nitrogen content of the feces. A moderate diarrhea has but little effect on absorption.

Previous Studies of Absorption in Typhoid.

Von Hösslin (7), in 1882, studied most carefully a series of typhoid patients fed on various diets, such as ham or milk or eggs, or the juice of pressed meat The total calories of his diets were not high and most of his patients suffered from diarrhea. The nitrogen of the food varied between 10 and 21 gm. and the feces nitrogen from 0.9 to 2.2 gm. or from 7.6 to 13 per cent. of the nitrogen ingested. With 50 to 135 gm. of fat in the food the feces contained from 5 to 10 gm. or from 6 to 10 per cent. The carbohydrates of the feces were not determined directly. Some of his patients were put on very low diets containing 1 to 3 gm. fat, 8 to 11 gm. carbohydrate and no protein. During these periods of practical starvation, the feces contained 0.8 to 5 gm. of ether extract a day and from 0.4 to 0.8 gm. of nitrogen. Von Hösslin came to the conclusion that foods were absorbed almost as well in typhoid fever as in health.

Shortly after his work was published the Russians of Chudnowsky's clinic, where typhoid patients were given liberal diets, began a series of investigation on the same subject. Most of their work is published in Russian and has never received the attention it deserves (8). Their work on the whole supports von Hösslin's contentions. They found the assimilation of protein to be almost as good as in health, and they found that cold baths, antipyretics, the drinking of water in large amounts and of alcohol in small amounts seemed to increase the percentage of protein absorbed. Large enemas of hot water seemed to decrease the absorption. Aikinov (8), who included in his dietary 20 gm. of blackberries, found from 4 to 6 gm. of nitrogen a day in the feces of his patients. Gruzdiev

(8), when he gave a very liberal diet of milk and bread with 30 to 45 gm. of nitrogen, found 4 to 11 gm. of nitrogen in the feces. The other observers using moderate and easily-digested diets obtained only 1 to 3 gm. per day. Chernoff (8), drawing his conclusions from a small majority of his cases, states that the assimilation of fat is better during the height of typhoid fever than during convalescence or health. Kurkutoff (8), on the other hand, found that the absorption of fat was poorer during fever and varied with the gravity of the disease.

On reviewing the figures of all these Russian investigators, one receives the impression that the absorption of food depends chiefly on the patient's general condition, and as this is improved either by the natural course of recovery or by therapeutic measures, the absorption of food improves also. It may be

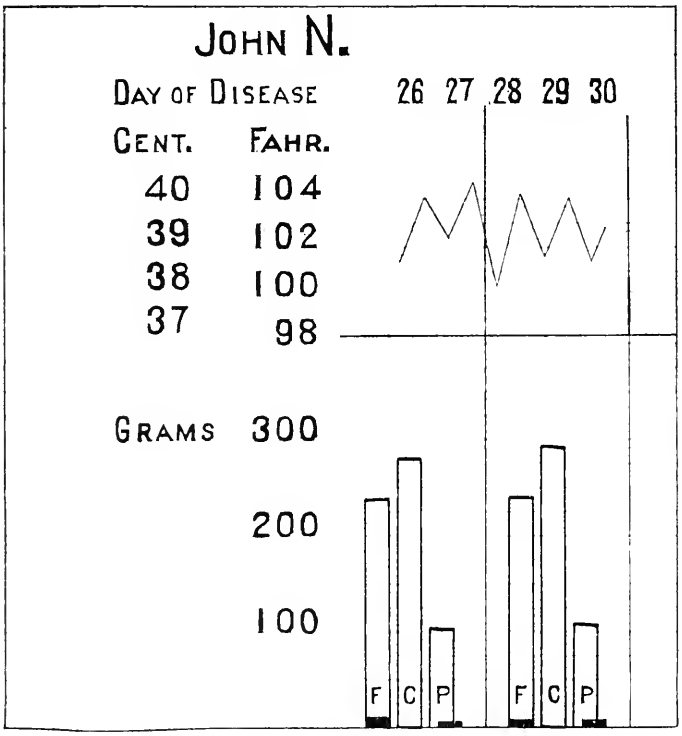

Chart 4.—Temperature curves, etc., in Case 4, John N.

noted in passing that the Russians, even when using liberal diets with very large amounts of protein, were unable to keep their fever patients in nitrogen equilibrium.

Von Leyden and Klemperer (9) were the next to study the absorption of foods incidental to their unsuccessful attempt to establish nitrogen equilibrium in typhoid. They found that patients with high fever when given 100 gm. of easily-digested fat lost from 6 to 11 per cent. in the feces, and lost about 9 per cent. of an equal amount of protein. Carbohydrates appeared in the feces only

when very large amounts were given in the food or when the patients suffered from profuse diarrhea.

Cases Studied.

The six typhoid cases on whom the present work was done were unselected cases from the wards of the second medical division (Cornell Division) of Belle-

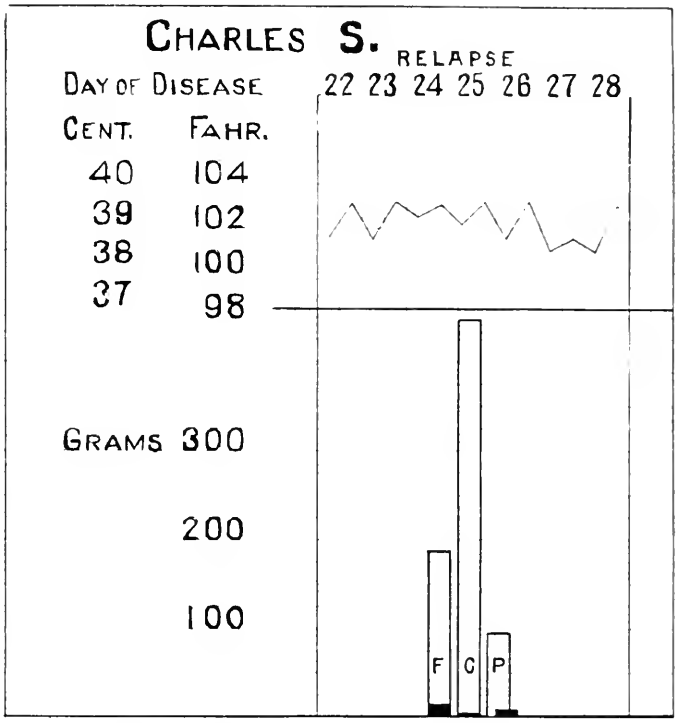

Chart 5.—Temperature curves etc., in Case 5, Charles S.

vue. It so happened that none of the patients had diarrhea during the period of investigation, but one is struck by the fact that patients able to take the high calory diet seldom have diarrhea. Many patients who enter the hospital with marked diarrhea begin to have normal stools after they have been for a couple of days on the diet. During the total of seventy-two days studied, there were only six stools in addition to the results of the daily enemas and none of these was diarrheal. The charcoal and carmin powder used to mark off the periods appeared in the feces twenty-four hours after being swallowed with such regularity that it was deemed safe in two cases to omit the line of demarkation between some of the periods. The daily enemas contained semi-formed, yellow feces of practically normal appearance and odor. In no case was there any blood in the stools. As routine the patients were given no medicine and no tubs, but were sponged for high temperatures.

Of the cases studied, three, Charles N., Michael K. and Frank W., were of rather mild type; three, Philip R., John N. and Charles S., were severe. It will be noted that a positive nitrogen balance was obtained in every case during periods when the temperature was still high. One has difficulty in stating when convalescence begins in such cases. As soon as the temperature starts to show

sharp morning remissions the patients look bright and seem comfortable. They read the newspapers, chat with their neighbors, eat their food with relish and rapidly gain weight and nitrogen-containing substances. Relapses are not more

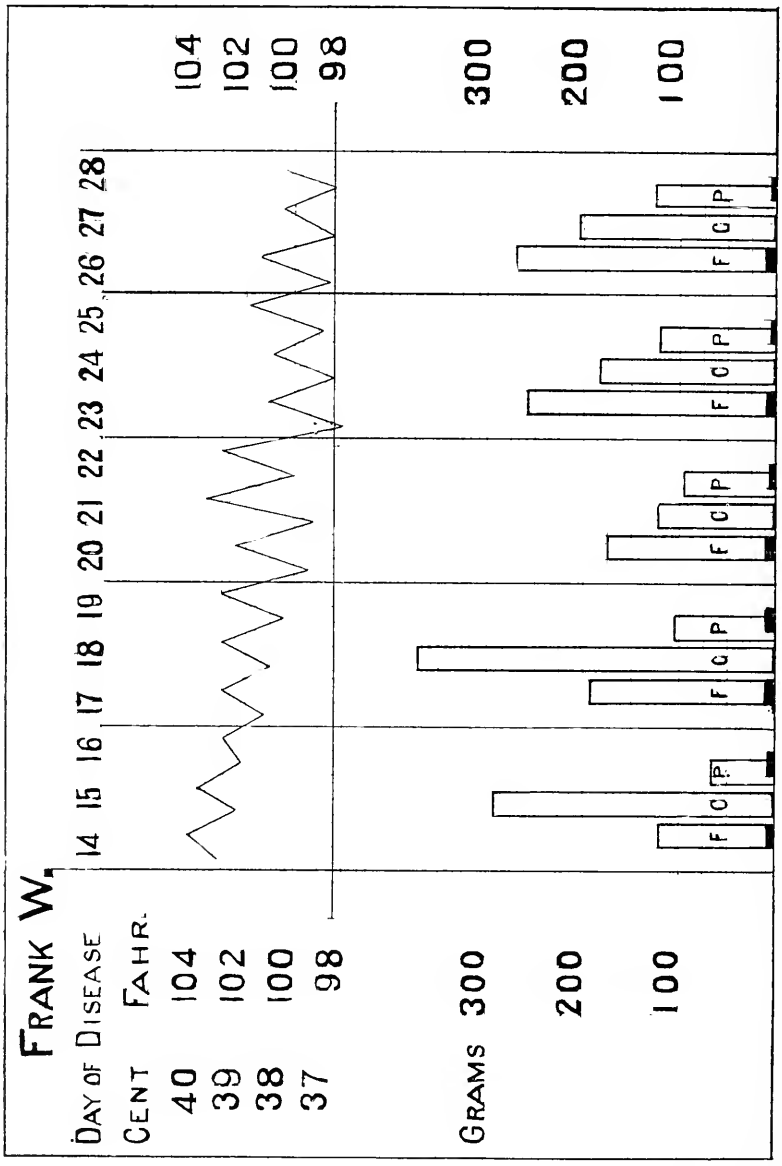

Chart 6.—Temperature curves, etc., in Case 6, Frank W.

frequent on the high diet than on low diets, and it is only chance that four of these six patients had relapses. The cases were as follows:

Case Reports.

Case 1.—Charles N., 60 years old, admitted Sept. 23, 1910, on the ninth day of the disease.

History.—The patient has been nursing three typhoid patients in his family. During the last four weeks he has been apathetic and at times dizzy. Date of onset of fever uncertain, probably nine days ago.

Physical Examination.—Well nourished elderly man. Right eye blind as a result of an old injury. Spleen palpable, several rose spots. Ninth to nineteenth day of disease. Temp. 101-103 F.; appetite good; takes 3,000 to 4,000 calories a day; no tympanites; mind clear; Widal test, negative. Nineteenth to twenty-first days, Period 1. Twenty-second to twenty-sixth days feces discarded because they were overheated when drying. Twenty-seventh to thirty-fourth days, patient improving steadily; thirty-fifth day patient has severe pain in the head. Three days later the pain localized in the right eye, which became much swollen. Diagnosis panophthalmitis. On the forty-first day the eyeball ruptured, discharging pus from which the typhoid bacillus was obtained in pure culture. By the forty-fifth day the eye was much improved and two days later the temperature reached normal. The patient made an uninterrupted convalescence.

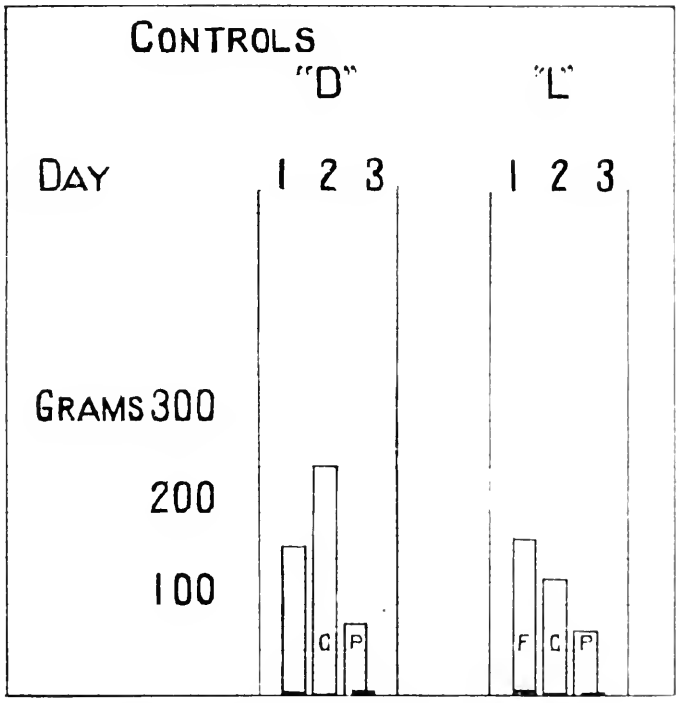

Chart 7.—Chart of control, Cases "D" and "L."

Case 2.—Michael K., 23 years old, admitted Oct. 13, 1910, on eighth day of disease.

History.—One week before admission the patient began to feel feverish and chilly and suffer from anorexia.

Physical examination.—Fairly well nourished young man; apathetic; marked tympanites; rose spots present. Eighth to thirteenth days, temperature 101-104 F. Blood culture shows typhoid bacilli. Appetite fair, takes 2,000 to 3,000 calories a day. No distention. Thirteenth day, feces collection started. Thirtieth day, temperature normal, convalescence rapid.

Table 2.—Metabolism Study in Charles N., Case 1

Day of Disease	Calories			Food Grams			Urine N + Feces N	Balance	Urine		Body Weight, Kilos
	Total	Per Kg.	Carbohydrates (per kg.)	Carbohydrates	Fat	Nitrogen			Vol., c.c.	Nitrogen	
19 (?) ..	4600	80	27	383	280	17.0	....				
20	3200	55	15	209	214	13.5	13.1	+ 0.4	1130	12.0	....
27	3910	68	16	226	268	19.2	....	.....	760	....	....
28	4030	70	17	245	272	19.3	13.2	+ 6.1	1260	11.8	....
29	4090	71	14	195	302	19.0	11.5	+ 7.5	890	10.1	....
30	3720	64	16	222	253	17.6	11.3	+ 6.3	1060	9.9	....
31	4040	70	20	279	261	18.9	15.0	+ 3.9	1520	13.6	57.7
32	4040	70	16	226	261	18.7	....	.....	1030	....	....
33	4670	81	22	314	311	19.2	12.3	+ 6.9	1290	11.1	....
34	3570	62	14	190	251	18.5	12.8	+ 5.7	1520	11.6	57.7
35	4410	76	19	261	305	19.5	14.6	+ 4.9	985	13.4	....
36	4300	75	16	223	312	19.8	11.8	+ 8.0	820	10.8	....
37	4520	78	19	266	312	20.8	13.3	+ 7.5	1415	12.3	....
38	4480	78	17	233	327	18.7	15.3	+ 3.4	1860	14.3	....
39	4060	71	16	227	284	19.2	15.2	+ 4.0	1500	13.4	57.5
40	4730	82	24	334	304	20.8	14.6	+ 6.2	1485	12.8	....
41	4420	77	18	247	311	20.1	12.0	+ 8.1	1200	10.2	....
42	4470	78	18	250	315	20.0	11.9	+ 8.1	1240	10.5	....
43	4170	72	18	253	274	22.7	....	.....	750	....	....
44	4490	78	17	245	314	21.9	10.4	+11.5	1100	9.0	57.5

Case 3.—Philip R., aged 23, admitted Oct. 19, 1910, on the seventh day of disease.

History.—For about three weeks the patient has felt chilly and feverish; headache severe; four to five waterly stools daily; took to bed six days ago.

Physical examination.—Fairly well nourished, pale, torpid, looks toxic; pulse small and weak; many rose spots; temperature 102-105 F.

Eighth to fifteenth days, experimental period: Twelfth day, pulse stronger, a few rales at bases of lungs; abdomen not distended. Thirteenth day, pulse weaker, patient given strychnin; slight distention. Seventeenth to twenty-second days temperature dropping to normal; patient feels better. Twenty-third to twenty-seventh days, temperature rising insteps to 105 F.; pulse weaker. Twenty-seventh to thirtieth days, temperature 102-105 F. Thirty-first to thirty-fifth days, experiment resumed. Thirty-seventh day, temperature normal. Convalescence slow.

Table 3.—Metabolism Study in Michael K., Case 2

Day of Disease	Calories			Food Grams			Urine N + Feces N	Balance	Urine		Body Weight, Kilos
	Total	Per Kg.	Carbohydrates (per kg.)	Carbohydrates	Fat	Nitrogen			Vol., c.c.	Nitrogen	
8	1970	35	8	115	134	9.6	....	.....	....	....	....
9	1970	35	8	115	134	9.6	....	.....	....	....	....
10	2540	45	10	133	176	12.2	13.1	— 0.9	1155	12.2	....
11	2540	45	10	133	176	12.2	19.1	— 6.9	845	17.9	....
12	2370	42	11	151	154	11.3	....	.....	Lost	....	....
13	2670	48	13	177	176	12.2	19.8	— 7.6	1940	18.6	....
14	3340	60	17	230	215	15.2	....	.....	Lost	....	....
15	4410	79	27	374	257	19.2	13.4	+ 5.8	1710	11.6	55.9
16	3960	71	25	323	242	14.4	18.2	— 3.8	1820	16.4	....
17	4790	86	31	427	257	20.2	17.6	+ 2.6	1200	16.0	....

Table 4.—Metabolism Study in Philip R., Case 3

Day of Disease	Calories			Food Grams			Urine N + Feces N	Balance	Urine		Body Weight, Kilos
	Total	Per Kg.	Carbohydrates (per kg.)	Carbohydrates	Fat	Nitrogen			Vol., c.c.	Nitrogen	
8	2400	44	9	123	166	13.9	....	.....	....	....	....
9	2580	48	11	148	179	11.8	....	.....	....	....	....
10	2470	45	11	143	175	12.1	16.8	— 4.7	1500	16.1	54.3
11	2830	52	14	186	179	15.8	17.2	— 1.4	1170	16.4	....
12	3100	57	13	178	215	15.3	19.7	— 4.4	1200	18.9	....
13	2890	53	14	180	191	14.5	20.8	— 6.3	1210	20.0	....
14	3460	63	17	230	225	17.2	14.9	+ 2.3	860	14.1	....
15	3810	70	16	214	268	17.9	14.9	+ 3.0	1340	14.1	54.7
31	2520	49	14	172	157	15.1	9.7	+ 5.4	900	8.8	51.8
32	2560	50	17	216	144	14.2	13.1	+ 1.1	1300	12.2	....
33	2900	56	19	242	160	17.6	13.9	+ 3.7	*	13.1	....
34	2880	56	26	310	135	14.4	9.5	+ 4.9	*	8.7	....
25	2920	59	20	245	159	18.2	14.0	+ 4.2	*	13.2	49.1

*Urine volumes made up to 2,000 c.c.

Case 4.—John N., aged 28, admitted Oct. 25, 1910, seventh day of disease. Died December 1.

History.—Onset six days ago with headache. Since then the patient has had anorexia and diarrhea.

Physical Examination.—Large frame, fairly well nourished, face flushed, abdomen flat, slight tenderness in left hypochondrium. A few rose spots were found.

Eighth day, blood culture shows typhoid bacilli. Tenth to eleventh days; slight soreness in abdomen and slight distention. Eighth to thirteenth days, temperature 101-104 F. Calories of food 2,500 to 3,500. Fourteenth to twenty-first days, temperature 100-103.6 F. Calories 3,600. Twenty-second to twenty-fifth days, temperature 102-105 F. Twenty-sixth to thirtieth days, period of investigation. Thirty-first to thirty-fourth days, temperature falling, patient more comfortable. Thirty-fifth day temperature shot up to 105 F. and a cough developed; during the next few days the left leg developed a boggy edema, the temperature remained high, signs of consolidation appeared in the left upper lobe. The patient became very toxic and died on the forty-fourth day of his illness.

Case 5.—Charles S. (service of Dr. C. L. Dana), aged 21, admitted Aug 5, 1911, fourth day of disease.

History.—Onset three days ago with chilly sensations and headache. Since then the patient has felt weak and feverish.

Table 5. Metabolism Study in John N., Case 4

Day of Disease	Calories			Food Grams			Urine N + Feces N	Balance	Urine		Body Weight, Kilos
	Total	Per Kg.	Carbohydrates (per kg.)	Carbohydrates	Fat	Nitrogen			Vol., c.c.	Nitrogen	
26	3540	55	16	256	227	17.0	11.0	+ 6.0	750	10.0	....
27	4030	62	19	301	246	17.4	15.7	+ 1.7	800	14.7	....
28	3870	58	18	280	245	18.2	18.0	+ 0.2	1100	17.1	....
29	3880	59	22	349	221	16.9	16.4	+ 0.5	850	15.5	....
30	3870	58	16	247	246	19.6	18.3	+ 1.3	1000	17.4	64.8

Table 6.—Metabolism Study in Charles S., Case 5

Day of Disease	Calories			Food Grams			Urine N + Feces N	Balance	Urine		Body Weight, Kilos
	Total	Per Kg.	Carbohydrates (per kg.)	Carbohydrates	Fat	Nitrogen			Vol., c.c.	Nitrogen	
22	2970	51	25	356.6	131.6	11.1	11.4	— 0.3	1255	10.2	....
23	4540	78	38	541.8	186|6	16.3	17.5	— 1.2	2350	16.3	58.2
24	5330	92	43	608.1	215.0	19.7	15.7	+ 4.0	1800	14.5	....
25	4240	73	33	463.5	205.0	16.8	12.5	+ 4.3	1650	11.3	....
26	2820	48	17	243.9	147.2	11.0	23,3	—12.3	2155	22.1	....
27	4450	77	33	465.5	195.0	15.4	14.5	+ 0.9	1110	13.3	....
28	4650	80	31	446.2	222.2	16.3	20.1	— 3.8	1220	18.9	58.2

Physical Examination.—Well nourished, abdomen flat, spleen palpable, a few rose spots.

Fourth to twelfth days temperature 101-103 F.; appetite good; not very sick. Seventeenth to nineteenth days, temperature normal. Twentieth to twenty-third days, temperature rising again in steps. Twenty-fourth to thirty-eighth days, sever relapse with temperature 102-104 F. On the twenty-fifth day a profuse nose bleed occurred, followed by several other severe attacks during the next two weeks. The patient became very anemic. The temperature fell slowly, reaching normal on the sixtieth day. Convalescence was slow.

The respiratory quotients of this patient and of the following case, Frank W., were investigated by Dr. Coleman and myself. The results will appear shortly.

Case 6.—Frank W., aged 27, admitted Nov. 23, 1911, on the tenth day of the disease.

History.—Nine days previously the patient began to have fever, headache and pains all over the body.

Physical Examination.—Small frame, 5 feet 4 inches tall, well nourished, prostrated, apathetic, spleen palpable, a few rose spots found.

Tenth to eleventh days, temperature 103-105 F.; appetite poor, diarrhea marked. Eleventh to thirteenth days, temperature 102 to 105 F.; diarrhea has ceased, appetite is improving. Fourteenth to twenty-eighth days, period of investigation. Appetite steadily improving; glucose was found in the urine in amounts which increased steadily until he passed 79 gm. on the nineteenth day. The carbohydrates of the food were then cut down until the sugar disappeared from the urine. The temperature fell steadily, reaching normal on the thirty-third day. The patient felt strong and was up in a chair when on the forty-seventh day from the onset the temperature began to rise and he went through a moderately severe relapse lasting fifteen days. On the seventh day of the relapse bilateral subconjunctival hemorrhages appeared but cleared up in a couple of weeks. Repeated urine tests during his rapid convalescence showed no sugar, although he was taking large amounts of carbohydrate.

Controls.—D. and L. These were two healthy young men between the ages of 25 and 30. They were given the typhoid diet but could not take as large amounts of food as the patients.

Table 7.—Metabolism Study in Frank W., Case 6

Day of Disease	Calories			Food Grams			Urine N + Feces N	Balance	Urine				Body Weight, Kilos
	Total	Per Kg.	Carbohydrates (per kg.)	Carbohydrates	Fat	Nitrogen			Vol., c.c.	Nitrogen	Glucose	Creatinin	
14	1910	35	21	276.0	67.2	5.9	19.9	—14.0	1222	18.5	Tr.	1.67	....
15	3380	62	25	333.7	169.2	16.9	16.6	+ 0.3	1210	15.2	7.72	1.28	54.5
16	2510	46	20	267.2	125.1	9.7	15.7	— 6.0	1052	14.3	8.03	1.27	....
17	3700	68	26	342.7	198.7	17.4	20.3	— 2.9	1455	18.9	15.48	1.35	....
18	3860	71	30	397.3	192.0	17.2	21.3	— 4.1	1675	19.9	31.6	1.59	....
19	3690	69	28	373.0	190.3	15.4	19.3	— 3.9	1800	17.9	79.6	1.29	53.6
20	3025	57	13	173.1	202.1	17.0	14.8	+ 2.2	1765	13.8	11.0	.99	....
21	2670	50	10	127.7	183.0	17.1	20.9	— 3.8	2050	19.9	8.5	1.17	....
22	1860	35	5	65.4	139.3	11.4	13.9	— 2.5	780	12.9	0.0	1.05	....
23	2860	54	10	131.4	200.3	17.9	19.7	— 1.8	1545	18.8	0.0	1.11	52.7
24	3980	76	16	201.5	282.4	20.5	17.4	+ 3.1	1575	16.5	0.0	....	....
25	4100	78	17	218.0	288.3	20.0	15.2	+ 5.4	1475	14.3	1.6	1.12	52.5
26	3750	71	15	195.3	264.7	19.0	12.7	+ 6.3	1300	11.9	1.64	....	....
27	3740	71	16	203.4	257.3	20.1	13.1	+ 7.0	1540	12.3	1.8	1.06	....
28	4100	76	16	215.0	287.6	21.1	18.2	+ 2.9	1790	17.4	1.6	1.29	53.9

Methods.

The patients were under the direct care of the head nurse, Miss Mary E. Sheehan, who has helped in metabolism experiments on typhoid cases for the last three years. All food given was measured and recorded. Samples of the milk and cream were analyzed from time to time and other foods were prepared according to known recipes and their food values calculated from the tables of Atwater and Bryant. (Bull. 28, U. S. Department of Agriculture.)

Every morning the nurse gave an enema of about 250 c.c. of warm water containing 0.75 gm. soap, which amount was, of course, subtracted from the fatty bodies found by analysis in the resulting stool. The results from these enemas were very uniform except in the case of Charles S., in whom an attempt was made to use salt solution instead of the usual soap enema. There was so much retention of feces in the lower bowel for the first three days that soap enemas were again resorted to and the period lengthened to seven days in order to get accurate results.

The periods were marked off at first by a teaspoonful of charcoal, which was somewhat difficult to recognize in the enemas. Later carmin powder in doses of 0.3 gm. was used and a most satisfactory line of demarcation obtained. In two of the earlier cases in which the food and stools were very uniform, the demarcation was omitted as the patients objected to charcoal.

As soon as the initial dose of charcoal was given, all urine was saved and all the feces as soon as the line of demarcation appeared. The enemas and feces were dried at a temperature below 100 C. with the addition of alcohol. The several stools of each period were then united, powdered, passed through a fine sieve and analyzed.

It was feared that the process of drying, which required one or two days, might cause a loss of some of the constituents. To determine this a normal man was put on the high calory typhoid diet and some of the formed stools thoroughly mixed and samples analyzed fresh and after drying in the above manner. The results show that the changes are negligible.

Table 8.—Analyses Before and After Drying, Expressed in Per Cent. of Moist Stool in Control Case.

	Fresh Feces	Dried Feces	Error Caused By Drying
Fat	4.20	4.38	.....
	4.34	4.43	.....
Average	4.27	4.405	+3.2
Carbohydrate	.97	.92	.....
	.97	.94	.....
Average	.97	.93	—4.0
Nitrogen	.829	.822	.....
	.874	.827	.....
	.860	.828	.....
Average	.8543	.8257	—3.3

All analyses were made in duplicate and if the results did not agree, were repeated until satisfactory. The nitrogen was determined by the Kjeldahl method, the fats by the complicated but exact method of Kumagawa and Suto (10), which determines the fats, fatty acids and soaps together. Results obtained by this method are usually higher than by the older methods of ether extraction which have been shown to be very faulty.

The carbohydrate determinations gave a great deal of trouble. It is impossible to make an accurate sugar test without decolorizing the feces, and many of the methods of decolorization remove sugar as well as color. The dried feces contained from 2 to 4 per cent. carbohydrates and after the processes hydrolizing and decolorizing, the remaining solution for analysis contained about .05 per cent. dextrose. Accurate sugar determinations with such dilute solutions are difficult.

Various methods of decolorization were tested, using the Allihn method, and the Pavy method as modified by Kumagawa and Suto (11): 0.25 gm. dextrose (Kahlbaum) was added to 4-gm. samples of a specimen of dried feces which gave no reduction after boiling with water, although it did reduce after hydrolizing with 2 per cent. HCl. The sample of feces with the added dextrose was boiled five minutes with 80 c.c. distilled water, cooled, 10 c.c. 20 per cent. HCl solution added and made up to 100 c.c. Two samples were filtered with difficulty, and Allihn determinations made with the highly colored filtrate. The others were decolorized by the mercuric nitrate method of Patein and Dufau (12) by basic lead acetate, mercuric bichlorid (13) and by acid charcoal.

Table 9.—Decolorization Tests of Feces in Control Cases

Decolorized by	Sugar Method	Per Cent. Error in Tests.
Mercuric bichlorid	Allihn	—11., +.09
Mercuric bichlorid	Pavy	+9.5, +2.6, —0.2
Mercuric nitrate	Allihn	+3.6, +5.4
Mercuric nitrate	Pavy	—10.5, —5.9
Basic lead acetate	Allihn	—7.2, —5.8
Basic lead acetate	Pavy	—7.9, —15.6
Acid charcoal	Allihn	—10.8, —5.8, +.08, +5.2, +2.8. Av. —1.7
Acid charcoal	Pavy	—11.0, —10.9, —5.1
Filtering; no decolorization	Allihn	—11, +0.9

It was clear that no method gave absolutely satisfactory results, but that the error of any method was not great enough to make a significant difference

in the findings. When one considers that many carbohydrate determinations in feces are made by the grossly inaccurate method of subtracting the total weight of fat, protein and ash from the weight of the dried stool, one becomes reconciled to a smaller acknowledged error. The method finally chosen for analysis was the acid charcoal method which seemed more accurate and simpler than any of the others. A specimen of 3 to 4 gm. of powdered feces was boiled one and one-half hours in 100 c.c. 2 per cent. HCl, in order to hydrolize the starches into sugars. After cooling, the solution was made up to volume, about 4 gm. of the best quality animal charcoal added, filtered, 2 gm. more charcoal added, filtered, an aliquot portion rendered slightly alkaline with NaOH to precipitate the phosphates, made up to volume and filtered. With 50 c.c. of the clear filtrate, Allihn tests were made. In a few cases, the modified Pavy method was used.

Soluble carbohydrates were tested for by boiling a similar sample of feces with water and acidifying after the solution had cooled. Charcoal was then used for decolorization and an Allihn test made. In no case was there more than a very slight reduction. It is an interesting fact that the feces of Michael K., who for the first two periods was given no carbohydrates except lactose, gave 0.4 and 1.2 gm. carbohydrate after hydrolizing, but none after plain boiling. Some reducing body or bodies other than carbohydrates must give this misleading result. Possibly mucin caused the reduction.

The indican was tested for in a roughly quantitative manner by Folin's method (14). One one-hundredth part of the total urine was treated with an equal volume of Obermeyer's reagent and the indigo blue extracted with 5 c.c. of chloroform and compared with Fehling's solution, which was given arbitrarily the value of 100. The color comparisons were made with Fehling's solution diluted to different percentages and were not made in a colorimeter. This method seems greatly preferable to the old method of recording the result in plus marks. Folin's normal individuals were on a diet very similar to the high calory diet and the indican excretion measured in this method ran between 12 and 140, the average of all six cases being 77.

Glucose in the urine was determined by Benedict's method (15), which gave most satisfactory results.

Summary.

Carbohydrates when given in amounts under 300 gm. a day were present in the stools only in traces, if, indeed, they were present at all. When amounts larger than 300 gm. were given, the stools sometimes contained 2 or 3 gm. of reducing bodies.

Table 10.—Comparison of Results.

Controls

	Fat			Carbohydrate			Nitrogen			
	Grams in Food	Grams in Feces	Per cent. Loss	Grams in Food	Grams in Feces	Per cent. Loss	Grams in Food	Grams in Feces	Per cent. Loss	Indican in Urine
"L"	172	6.4	3.8	127	.65	0.5	11.6	0.57	5.0	...
"D"	164	3.1	2.0	249	.25	0.1	12.7	1.00	7.8	...
Day of Disease.				**Charles N.**						
19-21	247	24.3	9.8	296	1.2	0.4	15.2	1.06	7.0	**100**
27-29	281	19.4	6.8	222	1.0	0.4	19.2	1.44	7.5	90
30-32	258	25.1	9.7	242	1.0	0.4	18.4	1.44	7.8	20
33-35	289	14.9	5.2	255	0.8	0.3	18.8	1.22	6.5	20
36-38	317	19.6	6.1	241	1.1	0.4	19.5	1.00	5.1	20
29-41	300	16.9	5.6	269	1.2	0.4	20.0	1.78	8.9	20
42-44	301	14.8	4.9	249	1.1	0.4	21.5	1.37	6.4	30
				Philip R.						
8-10	173	14.5	8.4	138	0.4	0.28	12.6	0.68	5.4	100
11-14	215	11.7	5.5	198	0.4	0.20	16.1	0.78	4.8	120
31-32	150	6.5	4.3	194	0.4	0.23	14.6	0.86	5.9	80
33-35	151	6.3	4.2	266	0.6	0.23	16.7	0.92	5.5	90
				Michael K.						
13-15	148	16.6	11.2	121	0.4	0.3	10.5	0.92	8.8	50
16-18	169	16.2	9.5	154	1.2	0.8	11.9	1.20	10.0	110
19-21	238	11.7	4.9	309	2.4	0.6	16.3	1.79	11.0	70
22	257	11.4	4.4	427	2.8	0.6	20.2	1.61	8.0	30
				John N.						
26-27	236	9.8	4.2	278	0.75	0.3	17.2	0.91	5.3	105
28-30	237	8.4	3.5	292	0.78	0.3	18.2	1.03	5.7	90
				Charles S.						
22-28	186.1	15.8	8.5	446.5	1.21	0.27	15.2	1.16	7.6	50
				Frank W.						
14-16	120.7	7.9	6.5	292.3	0.93	0.32	10.8	1.40	13.0	15
17-19	193.7	10.8	5.6	371.0	0.58	0.15	16.7	1.42	8.5	60
20-22	174.8	10.2	5.8	122.1	0.51	0.42	15.2	1.01	6.6	25
23-25	257.0	9.0	3.5	183.8	0.53	0.29	19.0	0.88	4.6	25
26-28	269.9	10.0	3.7	204.6	0.74	0.36	20.1	0.84	4.3	25

The nitrogen of the feces averaged 1.12 gm. a day, and never exceeded 1.8 gm. amounts which are within normal limits. The percentage loss was 7.1 per cent., which is a figure lower than that of previous observers. This, perhaps, may be due to the fact that the diet was less irritating to the intestinal tract.

With the fats there seems to be a diminution of both the percentage loss and the actual weight of fat in the feces as the disease progresses. It is hard to give averages which are fair, but it can be said that during the first three weeks of the attack and during the height of a relapse, the patients lose on an average 7.2 per cent. of the ingested fat. Later in the disease, with a falling temperature and decreasing toxemia, they lose about 4.5 per cent. The average loss for all cases examined was 6.02 per cent., which, though higher than the normal figure of 3 per cent. for a similar diet, is not enough higher to be of any clinical significance. The dried feces contained from 30 to 50 per cent. fat. It must be remembered that very large amounts of fat were given.

The stools of typhoid fever patients on the high calory diet resemble normal stools very closely. The indican of the urine, which is rather high during the early part of the disease, decreases steadily as the patient's condition improves. The indican excretion compares favorably with that of Folin's normal individuals (14).

The work of Shaffer and Coleman in establishing nitrogen equilibrium in typhoid fever has been confirmed.

Conclusions.

Typhoid patients throughout the disease can absorb carbohydrates and protein as well as normal individuals. They can absorb very large amounts of fat, but the percentage of absorption is somewhat lower than the normal, especially in the earlier part of the disease.

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THE BACTERIOLOGY OF SPUTUM IN COMMON NON-TUBERCULOSIS INFECTIONS OF THE UPPER AND LOWER RESPIRATORY TRACTS. WITH SPECIAL REFERENCE TO LOBAR AND BRONCHO-PNEUMONIA.*

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In a concise and interesting paper, Kitasato (1) discusses the bacteria in the sputum from pulmonary tuberculosis and gives the simplest and most reliable method of isolating bacteria from sputum containing several organisms. The cultural investigations here recorded were stimulated by the paper of Kitasato and, in 1905, by that of Schottmüller (2); and the article of Norris and Pappenheimer (3) called our attention to the fact that post-mortem bacterial examinations of material from the respiratory tract are not reliable, and showed that careful sputum examinations might be of considerable value in prognosis and also in treatment, now that specific therapy is being developed.

The examinations of infections of the respiratory tract here recorded are for the years 1903 to 1910, and were made upon patients visiting the Dispensary of Cornell University Medical College, upon those admitted to the wards of the Second Division of Bellevue Hospital, and upon a few private patients. The earlier examinations were made by Dr. Hastings and Dr. Mortimer Warren, the later ones by Dr. Armstrong and Dr. Niles. The study of the sputum throughout a series of years has shown a marked yearly variation in the types of infecting organism; so that it is safe to conclude that the observations on one set of cases for a few months have little weight in the determination of the infectivity of an organism for the respiratory tract and of the frequency of the association of any one germ with certain clinical symptoms and signs.

The Value of the Literature Upon the Bacteriology of the Respiratory Tract.

The literature regarding the bacteriology of the healthy respiratory tract below the larynx is voluminous, as the subject has been extensively investigated in man and animals. Nevertheless, in most of the work two serious errors are found which vitiate the strength of the conclusions.

The first error is, that cultures have usually been taken post-mortem, and the findings assumed to represent the bacterial flora ante-mortem. That this is fallacious was shown by the investgations of von Besser (4), and more recently by Norris and Pappenheimer who state: "It follows logically from the results obtained in this experiment that the cultural findings after death are no guide to the bacterial contents of the lungs during life, and that any deductions made from such findings are unreliable and deceptive."

The second error is self-evident and consists in drawing conclusions from examinations of sputum that has been collected without regard to the contami-

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nation that must take place unless precautions are observed while the sputum is passing through the pharynx and mouth.

It seems to have been generally assumed that the bronchi and alveoli are normally infected, and such authorities as Baumgarten (5) and Hoffmann (6) state that such is the case. Baumgarten, however, offers no proof, and Hoffmann merely quotes Pansini as having found several species of bacteria in healthy lungs, but gives no details. Dürck (7), who examined healthy lungs of man and animals, is quoted as believing in normal lung infection, but as his work was done post-mortem, his conclusions can not be accepted without reservation.

Results of Earlier Investigations in Special Diseases.

It seems hardly worth while to review much of the literature on the bacteriology of respiratory diseases because of the variations in technique and the lack of precautions observed by the investigators. Some of the more recent and important work will, however, be noted.

In acute catarrhal inflammation of the nose, White (8) reports that B. coryzae segmentosus (1) was present in fifty out of fifty-six cases examined, and he regards it as the most frequent cause. At the same time (1906), Allen (9) apparently regarded the bacillus of Friedländer as more important. In 1908, however, he reported the bacteriology of forty-two "colds" occurring in London during the previous three years. This time he found B. influenzae alone in 2.4 per cent., B. Friedländeri in 19.0 per cent., B. coryzae segmentosus in 26.2 per cent., and Micrococcus catarrhalis in 28.6 per cent. of the cases. He concluded that any of the above mentioned organisms might cause acute nasal catarrh; that subacute catarrh is commonly caused by the bacillus of Friedländer or by Micrococcus catarrhalis, but rarelly by B. influenzae, or by B. coryzae segmentosus, and chronic catarrh by Friedländer's bacillus only.

In tracheal catarrhs, Allen (10) concludes that B. influenzae or Micrococcus catarrhalis are usually found, the bacillus of Friedländer exceptionally. Micrococcus paratetragenus was not found by him, although it has been reported in trachitis by Benzançon and De Jong (11), and by Benham (12).

As inflammation of the bronchial mucous membrane often accompanies specific diseases and often arises independently, it seems well, as Marfan (13) has suggested, to adopt a bacteriological classification. Marfan makes two general groups. Group 1 is due to specific infections (influenza, pertussis, measles, diphtheria, anthrax, plague, tuberculosis, variola, malaria, glanders, and syphilis), and group 2 is due to non-specific infections. These he believes arise chiefly from pneumococci and streptococci.

B. influenzae is constantly found in the bronchitis of epidemic influenza, and Kretz (14), Washbourn (15), and Lord (16) have called attention to its frequency in the respiratory tract in diseases other than epidemic influenza. Davis (17) and Wollstein (18) report an organism, which appears to be identical with the influenza bacillus, as almost constantly present in the sputum of pertussis. Goldie (19) has seen two fatal cases of general bronchitis in which no membrane formed, but from the secretions of which pure cultures of the diphtheria bacillus were obtained.

Many forms of microorganisms have been found in exudates of non-specific bronchitis, and the infection is usually a mixed one, two, or three, or more varieties being present. Undoubtedly some are often secondary invaders, but pathogenic properties have been attributed to many.

Pneumococci and streptococci alone, or associated with each other or with

1. We have found B. coryzae segmentosus once in the sputum of chronic bronchitis in a dog.

other bacteria, have been most frequently reported. Ritchie (20, 21), who has thoroughly reviewed the literature of the subject, examined forty-nine cases and found these two organisms most commonly. His work, however, like that of many other investigators, is open to the criticism that his cultures, though made from the smaller bronchioles, were taken post-mortem, and may, therefore, be misleading. Among those who found pneumococci predominating are von Besser (22) and Barthel (23); while Kruse, Pansini and Pasquale (24), Bouchard (25), Claisse (26), Queyrat (27), Cassaët (28), Hoffmann (29), and Forchheimer (30) found streptococci in abundance. Babes and Popesco (31) regard Staphylococcus aureus as more important, and Durante (32) reports staphylcocci in pure culture in four cases. Patton (33) found streptococci and staphylococci in most cases.

An important recent report is that of Pollak (34), who examined seventy-three cases and found twelve varieties of bacteria, including B. coli communis, B. lactis aerogenes, and B. proteus.

Ghon and Pfeiffer (35) and Sederl (36) have emphasized the importance of Micrococcus catarrhalis in acute bronchitis. Others which seem occasionally to be the specific cause are, B. Friedländeri, Micrococcus tetragenus, and several saprophytes.

The frequency of B. influenzae in bronchiectasis has been emphasized by Boggs (37) who reports five cases, in three of which the bacilli were in pure culture, and present with pneumococci in the other two cases. Sections from two fatal cases revealed B. influenzae deep in the walls of the bronchi and mostly intracellular.

Lobar (Croupous) Pneumonia.—The Diplococcus lanceolatus (pneumococcus) has been so constantly demonstrated in the exudate of acute lobar pneumonia that it is generally regarded as the specific etiologic agent. This view receives corroboration by the demonstration of an accompanying pneumococcemia in a large proportion of cases. Prochaska (38) having found it in all of fifty cases, and Rosenow (39) in 132 out of 145 cases, both believing the blood invasion to be a constant condition. It is probable that other organisms may occasionally induce a pneumonia which can not be clinically or pathologically differentiated from that caused by the pneumococcus.

This property has been long attributed to Friedländer's bacillus, and Schottmüller (2) has described five fatal cases of apparently typical lobar pneumonia, also one non-fatal empyema from which he recovered Streptococcus mucosus in pure culture. Secondary invasion by other microorganisms is common, and it seems likely that these may at times influence the course of the disease.

Streptococcus pyogenes is regarded as the most common associate of the pneumococcus, although Staphylococcus pyogenes aureus, B. influenzae, B. diphtheriae, and Micrococcus catarrhalis are not infrequent.

Broncho-Pneumonia.—In this form of pneumonia no specific organism has been determined. The infection is usually a mixed one, but in the primary form of pneumococcus alone or associated with others is the bacterium found most frequently. Wollstein (40) took cultures post-mortem from infants and found pneumococci in 81 per cent. of thirty-three cases, and in pure culture in 41 per cent. Two cases showed streptococci alone. Staphylococcus aureus was recovered in pure culture from two cases, and in one case it was found in association with B. coli communis.

Pfeiffer (35) and Sederl (36) report Micrococcus catarrhalis in pure culture from broncho-pneumonia as well as from bronchitis.

In the secondary form, mixed infections are again the rule, all the above mentioned organisms having been found in association with specific cause of the primary disease.

Sterility of Upper and Lower Respiratory Passages.—In order that the bacteriological findings in exudates from the respiratory tract may be of value in representing the specific cause of an inflammation, it is necessary to establish the validity of two propositions: (1) that most of the normal respiratory tract, particularly its lower part, is sterile, and (2) that it is possible to collect and handle exudates in such a manner that bacterial contamination from the normally infected parts may be obviated.

The Nasal Mucosa.—The evidence regarding the sterility of the healthy nasal mucosa is conflicting. This is principally because of the difficulty in deciding whether or not a nose is really normal, but partly because every precaution may not have been taken in collecting exudates. All agree that large numbers of atmospheric bacteria are constantly found in the vestibula of the nares and on the vibrissae, which act as filters, and contamination from these sources is likely, unless precautions are observed. The careful work of Fraenkel (41) and Löwenberg (42), who found no bacteria in the interior of healthy noses, is, however, convincing. Others who have found most healthy noses sterile are Hildebrandt (43), Lermoyez and Wurtz (44), and Thomson and Hewlett (45). Lewis and Turner (46) conclude that healthy accessory sinuses are also probably sterile.

The Mouth and Pharynx.—The mouth and pharynx, whether healthy or diseased, are, of course, commonly infected with the atmospheric microorganisms, and not infrequently with bacteria, which are commonly pathogenic, notably pneumococci (Hiss, 47).

The Respiratory Tract Below the Larynx.—Many observers, however, have found the air passages of healthy individuals from the glottis down to be usually sterile, or at most to contain very few bacteria. Among these may be mentioned Babes (48), Thomson and Hewlett (49), Muller (50), Barthel (23), Klipstein (51), and Jundell (52).

Jundell carried out a particularly interesting investigation, obtaining mucus from the tracheas of forty-three healthy human beings by means of a special instrument which he devised. That those observations are probably correct is borne out by the following conclusions of Ritchie (20):

(a) All bacteria contained in inspired air are probably withdrawn in the winding upper air passages.

(b) The nasal mucous membrane possesses marked bactericidal properties.

(c) Inspired air remains mostly in the upper portions of the bronchial tract, seldom reaching the alveoli.

(d) Expired air contains no bacteria.

(e) Bacteria when introduced into the bronchi or lungs of healthy animals soon die.

We agree with Krehl (53) and Ritchie (20), both of whom conclude that the air passages of healthy individuals below the glottis are usually sterile.

Exudates formed in portions of the respiratory tract that are normally sterile may be collected and treated in a way that will prevent contamination. The anterior nares are readily disinfected and this should be done before collecting exudates from the nose. Contamination in the mouth and pharynx is avoided by the method of Kitasato (1) who collected in a sterile dish sputum

from deep coughing, after which he picked a small piece from the center, washed it in sterile water, and then planted it on proper media. If previous to this collection the mouth and pharynx are washed with salt solution or a mild antiseptic, there is little liability of contamination. The more elaborate method of Löwenstein (54) seems unnecessary (2).

We assume, therefore, that when the proper precautions are observed, the bacteria found in an exudate from the respiratory tract are the specific cause of the inflammatory process which produced the exudate.

Methods Employed and Nomenclature Adopted.

The technique used was that suggested by Kitasato. The mouth and pharynx were rinsed with sterile water or salt solution and then the sputum from a single expectoration was collected in a bottle that had been sterilized by boiling. From the sputum thus obtained the portion to be examined was selected and washed in 0.8 per cent. sterile salt solution.

A clean sputum, i. e., one containing only two or three types of bacteria and free from buccal squamous cells, and a dirty sputum, i.e., one containing a varied bacterial and fungoid flora and buccal squamous cells, are readily recognized on microscopical examination.

A dirty sputum is not suitable for bacterial examination and should be discarded for a second or third clean specimen from the same patient.

The more carefully the clean specimens are selected, washed, and handled, the more frequently does one obtain pure, unmixed cultures. If collected and handled properly, washing may be dispensed with. Inoculations should be made on tube-slants of plain and glycerine agar and in plain broth, and from the first tube of broth a platinum loop full or two should be transferred to a second tube of broth, from which streaks should be made upon agar plates. Except when Micrococcus catarrhalis is present, a mixed flora may, as a rule, be easily separated on the first trial. For differentiation, the usual bacteriological methods are to be followed, namely, the making of cultures upon glucose and other carbohydrate media, upon hemoglobin media, and upon blood serum. Rarely, anaerobic cultures may be necessary.

Whenever the stained smears from the sputum have shown small gram negative bacilli which might be Bacillus influenzae, cultures have been made directly upon fresh hemoglobin media.

Nomenclature.—The term micrococcus has been used for the various cocci, including staphylococci, e. g., Micrococcus aureus, Micrococcus albus, Micrococcus catarrhalis. Streptococci (3) have been classed, wherever the proper differentiation has been carried out, as Streptococcus pyogenes (longus et erysipelatous), Streptococcus hemolysans (longus seu brevis), Streptococcus viridans or mitior (longus seu brevis).

The Streptococcus mucosus and pneumococcus have been considered as more closely related to each other than to the three types of streptococci mentioned.

The bacillus of Friedländer and the other types of closely related gram negative, encapsulated, aerobic bacilli have been classed under Bacillus mucosus capsulatus.

The organisms found were as follows: Streptococcus pyogenes, Strepto-

2. Lowenstein collected in a sterile dish early morning sputum after having the patient wash the mouth with a thymol mouth-wash after meals and at night, for the three preceding days. He then washed the pieces of sputum in hot water and followed this with a quick washing in a 3 per cent. solution of hydrogen peroxid.

3. Schotmüller (55) classifies the three groups of streptococci as: (1) Streptococcus longus et erysipelatous, (2) Streptococcus mitior seu viridis, and (3) Streptococcus mucosus.

coccus hemolysans and Streptococcus mitior, pneumococcus, Micrococcus catarrhalis, Micrococcus tetragenus, Micrococcus aureus, Micrococcus albus, Micrococcus citreus, Streptococcus mucosus, Bacillus mucosus capsulatus (which includes Bacillus Friedländeri), Bacillus influenzae, Bacillus fluorescens, Bacillus pyocyaneus, Bacillus coli, and Bacillus acidi lactici.

Groups of Non-Tuberculous Diseases Studied.

Our 183 cases have been divided into nine groups. The first two of these deal with the upper respiratory tract, the larynx, trachea, and larger bronchi, and the other seven with the lower tract, the smaller bronchi, vesicles, and pleurae.

Group 1 consists of twelve cases; i. e., acute laryngitis (2), acute trachitis (9), and chronic trachitis (1).

Group 2 contains twenty- seven cases of grippe, nineteen of these with or without coryza (including influenza), and eight with no diagnosis other than grippe, probably acute bronchitis (including influenza).

Group 3 embraces nine cases of acute bronchitis.

Group 4 consists of forty-eight cases of chronic bronchitis.

Group 5 embraces sixty-six cases of lobar pneumonia. In only twenty-three of these were pneumococci found in culture.

Group 6 includes twelve cases of bronchial pneumonia; three were typical, and nine atypical, the so-called grippe or influenza pneumonia.

Group 7 contains two cases of bronchiectasis.

Group 8 consists of two cases of asthma.

Group 9 includes five cases of pleuritis, or pleurisy, with no other diagnosis.

Summary of Cultures From Glottis Down, in Order of Frequency.

Organisms	No. of positive sputa.	Percentage of occurrence.
M. catarrhalis	71	21.0
M. aureus	52	15.0
Streptococci	50	14.0
Pneumococcus	45	13.0
		—63.0
M. tetragenus	35	10.0
M. albus	21	6.0
B. Friedländeri	13	4.0
B. influenzae	6	1.5
		—21.5
Streptococcus mucosus	5	1.5
M. citreus	4	1.25
B. fluorescens	4	1.25
		— 4.0
B. coli	3	1.0
B. pyocyaneus	3	1.0
B. proteus	3	1.0
Oidium albicans	2	—1.0
Hay bacillus	2	—1.0
B. acidi lactici	1	+ .5
		— 5.5

Unidentified organisms	11	3.0
Total No. of isolations—	331	—97.0
Sputa culturally negative	10	3
Total No. of cultures—	341	100

Summary of Cultures by Year.

	Year.	Number.	Most common.	Number of times isolated.	Per cent.
1.	1903–04	34	Pneumococcus	8	23.5
			M. catarrhalis	7	20.5
			M. aureus	5	14.5
			M. tetragenus	3	9.0
			M. albus,	3	9.0
			Other organisms and negative cultures	8	23.5
2.	1904–05	33	M. catarrhalis,	18	29.0
			Streptococci	9	14.0
			M. tetragenus	8	13.0
			Pneumococcus	6	9.5
			M. aureus	6	9.5
			Other organisms and negative cultures	16	25.0
3.	1905–06	31	M. catarrhalis	5	16.0
			M. tetragenus	**4**	**13.0**
			M. aureus	4	13.0
			Pneumococcus	3	10.0
			Other organisms and negative cultures	15	48.0
4.	1906–07	23	M. tetragenus	5	22.0
			Streptococci	4	17.5
			Pneumococcus	3	13.0
			Streptococcus mucosus	3	13.0
			Other organisms and negative cultures	8	34.5
5.	1907–08	84	M. catarrhalis	17	20.5
			Streptococci	16	19.5
			M. tetragenus	12	14.0
			Pneumococcus	12	14.0
			Other organisms and negative cultures	27	32.0
6.	1908–09	55	Streptococci	12	22.0
			Pneumococcus	10	19.0
			M. catarrhalis	9	17.0
			M. aureus	8	15.0
			Other organisms and negative cultures	16	27.0
7.	1909–10	51	M. aureus	18	35.5
			M. catarrhalis	15	29.5
			Streptococci	6	11.5
			Pneumococcus	3	6.0
			Other organisms and negative cultures	9	17.5
Total number of cultures				341	

The Organisms Present in the Clinical Groups.

Group 1 (Laryngitis and Trachitis).—The organisms most frequently isolated were: Micrococcus catarrhalis (7 times) and Micrococcus aureus (6 times). Pure cultures of Micrococcus aureus were obtained in 3 cases, and Micrococcus catarrhalis in 1.

Group 2 (Grippe and Influenza, probably with Acute Bronchitis).—In this group Micrococcus catarrhalis was the organism most frequently isolated (15 times), streptococci (4) were found in 7 cases, and the pneumococcus in 7. Bacillus influenzae was present in 7, and Micrococcus tetragenus in 6 cases. In 8 instances the sputa gave pure cultures: Micrococcus catarrhalis in 4, Micrococcus albus in 2, Micrococcus tetragenus in 1, and streptococcus in 1.

Group 3 (Acute Bronchitis).—Micrococcus catarrhalis was the organism most commonly found, being isolated 6 times. Two of the cases gave pure cultures, one of Micrococcus catarrhalis and one of pneumococcus.

Group 4 (Chronic Bronchitis).—The organisms isolated from this group and their frequency were as follows: Micrococcus aureus (17), streptococci (15), Micrococcus catarrhalis (13), Micrococcus tetragenus (10), pneumococcus (18), Streptococcus mucosus (4), Bacillus influenzae (1). Pure cultures were obtained in 18 cases, as follows: Micrococcus aureus (7), Micrococcus albus (2), pneumococcus (2), Micrococcus catarrhalis (1), streptococci (1), Streptococcus mucosus (1), Micrococcus tetragenus (1), Bacillus coli (1), Bacillus pyocyaneus (1), Friedländer's bacillus (1).

Group 5 (Lobar Pneumonia).—There were twenty-three cases with pneumococci and forty-three cases with no pneumococci in the sputum. These results do not represent the proportion of cases in which the pneumococcus occurs in lobar pneumonia, for many of the cultures were taken because the clinical manifestations of the disease were atypical.

Of the twenty-three sputa giving pneumococci, nine revealed them in pure culture. The organisms were as follows: Micrococcus catarrhalis in 7 cases, streptococci in 3, Micrococcus aureus in 3, Micrococcus tetragenus in 5, Micrococcus albus in 5, Friedländer's bacillus in 1, Bacillus proteus in 1, Micrococcus citreus in 1, and unidentified organisms in 6.

From the forty-three sputa without pneumococci in cultures, the following organisms were most frequently recovered: streptococci in 14, Micrococcus catarrhalis in 16, Micrococcus aureus in 11, Micrococcus tetragenus in 8, Friedländer's bacillus in 4, and Bacillus coli in 2 cases. The pure cultures from these cases were 18 in number, as follows: Micrococcus aureus in 6, streptococci in 5, Micrococcus catarrhalis in 4, Bacillus coli in 1, Bacillus proteus in 1, and Micrococcus citreus in 1.

Fresh smears were made from every exudate, and in only four of the above were capsulated cocci in pairs observed. In four of the negative cases the pneumococcus was found neither in cultures nor smears. In three others the cultures were reported negative for pneumococci and smears were positive for pneumococci. We consider that a few scattered pneumococci might be found in smears from buccal cavity contamination, while the cultures might be negative. The opposite of this, i. e., that smears might be negative and cultures positive, is untenable, and our results bear this out. The hay bacillus in one case was a contamination.

From lobar pneumonia the most common isolations were: the pneumococcus in 23 instances, Micrococcus catarrhalis in 23, streptococci in 17, Micrococcus aureus in 14, Micrococcus tetragenus in 13, Micrococcus albus in 7, Bacillus Friedländeri in 5, Bacillus proteus in 2, Bacillus coli in 2, Bacillus fluroscens in

4 The types of streptococci have not been separated for this report. From a study of the types isolated from specimens in this laboratory they have been classed as (a) Streptococcus pyogenes (longus et erysipelatous), (b) Streptococcus hemolysans (longus seu brevis), and (c) Strentococcus viridans seu mitior (longus seu brevis). Our classification is a modification of that of Schottmüller.

1, Micrococcus citreus in 3, and unidentified organisms in 6. The total number of isolations from lobar pneumonia was 110.

Group 6.—In broncho-pneumonia the organisms most frequently isolated were: Micrococcus catarrhalis in 5 cases, streptococci in 4, Micrococcus aureus in 14, pneumococcus in 2, and Bacillus Friedländeri in 2. Pure cultures were obtained from seven cases, as follows: Micrococcus aureus from 23, Bacillus Friedländeri from 2, Bacillus pyocyaneus from 1, and pneumococcus from 1.

Group 7.—One exudate from bronchiectasis revealed a pure culture of the streptcoccus, and from the other were isolated Micrococcus albus and Bacillus proteus.

Group 8.—From the bronchial asthma group, Micrococcus catarrhalis and the Micrococcus tetragenus were each isolated once in pure culture.

Group 9.—The five cases of pleurisy in this group could not be proven tuberculous. The production of sputum is evidence of some condition other than pleurisy, but no other diagnosis appeared in the records. The pneumococcus was isolated in 3 cases; the micrococcus in 2; Bacillus Friedländeri in 1; and Bacillus fluorescens in 1. One case gave a pure culture of pneumococcus.

The yearly variation is noteworthy, and also the fact that it can not be accounted for by variations in the number of examinations made in the different years. The organisms which were first in frequency, and the winter seasons from 1903 to 1910 inclusive during which they were first are as follows: Micrococcus catarrhalis (1904-05), Micrococcus catarrhalis (1905-06), Micrococcus catarrhalis (1907-08), pneumococcus (1903-04), streptococci (1908-09), Micrococcus aureus (1909-10), Micrococcus tetragenus (1906-07); and those second in frequency were: pneumococcus (1908-09), streptococci (1904-05), streptococci (1906-07), streptococci (1907-08), Micrococcus catarrhalis (1903-04), Micrococcus catarrhalis (1909-10), Micrococcus tetragenus (1905-06).

Summary.

1. In our examinations, only 38 per cent. of the infections of the respiratory tract below the glottis were pure, and this percentage was reached only by carefully following Kitasato's method of handling sputa.

2. Lobar pneumonia may produce sputum free from pneumococci, and may undoubtedly be caused by organisms other than the pneumococcus.

3. There is found a marked yearly variation in the organisms which excite inflammation of the respiratory tract.

4. Micrococcus catarrhalis is usually considered a common secondary invader; but it may, and probably frequently does, assume pathogenic properties.

We extend our appreciation particularly to Dr. Thomas H. Evans for his valuable assistance in verifying the records and compiling the tables.

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Group numbers.	Groups and subdivisions.	No. of cases.	Year.	Streptococcus.	Pneumococcus.	M. catarrhalis.	M. tetragenus.	M. aureus.	M. albus.	M. citreus.	Streptococcus mucosus.	B. mucosus capsulatus.	B. influenzæ.	B. fluorescens.	B. proteus.	B. pyocyaneus.	B. coli.	B. acidi lactici.	Unidentified.	Oidium.	Pneumococcus in smears.
I	Laryngitis (acute)	1	1903–04	....	+	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1905–06	+	....	+	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....
I	Trachitis (chronic)	1	1909–10	+	....	+	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....
I	Trachitis (acute)	1	1904–05	....	....	+	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....
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I	Laryngitis (acute)	1	1903–04	....	+	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1905–06	+	....	+	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....
I	Trachitis (chronic)	1	1909–10	+	....	+	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....
I	Trachitis (acute)	1	1904–05	....	....	+	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....
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		1	1904–05	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....
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V	Pneumonia (lobar, with no pneumococci in sputum)	1	1904–05	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1904–05	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
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		1	1906–07	+	....	....	....	....	....	....	....	....	+	....	+	....	....	....	....	....	+
		1	1906–07	+	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	+
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		1	1907–08	....	....	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....
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B.

TABULATED SUMMARY OF ISOLATIONS ACCORDING TO SEASON AND INCIDENCE.—CONTINUED.

Group numbers.	Groups and subdivisions.	No. of cases.	Year.	Streptococcus.	Pneumococcus.	M. catarrhalis.	M. tetragenus.	M. aureus.	M. albus.	M. citreus.	Streptococcus mucosus.	B. mucosus capsulatus.	B. influenzæ.	B. fluorescens.	B. proteus.	B. pyocyaneus.	B. coli.	B. acidi lactici.	Unidentified.	Oidium.	Pneumococcus in smears.
V	Pneumonia (lobar, with no pneumococci in sputum)	1	1904–05	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1904–05	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1905–06	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1905–06	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1906–07	+	....	....	....	....	....	....	....	....	+	....	+	....	....	....	....	....	+
		1	1906–07	+	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1907–08	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1907–08	+	....	+	+	....	+	....	....	....	....	....	....	....	....	....	....	....	+
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		1	1907–08	+	....	....	+	....	+	....	....	....	....	....	....	....	....	....	....	....	+
		1	1907–08	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
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		1	1907–08	+	....	....	....	+	....	....	....	....	....	....	....	....	....	....	B	....	+
		1	1908–09	....	....	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1908–09	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1909–10	....	....	+	....	....	....	....	....	....	....	....	....	....	+	....	....	....	+
		1	1909–10	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1909–10	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
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		1	1909–10	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
V	Pneumonia (lobar, with pneumococci in sputum)	1	1903–04	....	+	+	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1903–04	....	+	+	....	....	+	+	....	....	....	....	....	....	....	....	....	....	+
		1	1904–05	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1906–07	....	+	....	....	....	+	....	....	....	....	....	....	....	....	....	....	....	+
		1	1907–08	....	+	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1907–08	....	+	+	+	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1907–08	....	+	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
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		1	1907–08	....	+	....	....	....	+	....	....	....	....	....	....	....	....	....	....	....	+
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		1	1909–10	....	+	....	....	....	+	....	....	....	....	....	....	....	....	....	....	....	+
VI	Pneumonia (bronchial, typical)	1	1904–05	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+	....	....	+
		1	1908–09	+	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1908–09	....	....	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	+
VI	Pneumonia (bronchial, atypical)	1	1903–04	....	....	....	....	....	....	....	....	....	+	....	....	....	....	....	....	....	....
		1	1904–05	....	....	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1905–06	....	....	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....
VI	Pneumonia (bronchial, atypical, or typhoid)	1	1904–05	....	+	+	+	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1905–06	....	....	....	....	....	....	....	....	....	+	....	....	....	....	....	....	....	+
VI	Pneumonia (bronchial, atypical, and sepsis)	1	1908–09	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1907–08	+	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1908–09	+	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	+
		1	1908–09	+	....	+	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....
VII	Bronchiectasis	1	1904–05	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1907–08	....	....	....	....	....	+	....	....	....	....	....	+	....	....	....	....	....	....
VIII	Asthma	1	1903–04	....	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1904–05	....	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....
IX	Pleuritis (with no other diagnosis)	1	1905–06	....	....	+	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....
		1	1905–06	....	....	+	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....
IX	Pleurisy (with emphysema)	1	1904–05	+	....	....	....	....	....	....	....	....	+	....	+	....	....	....	....	....	....
IX	Pleurisy (with effusion)	1	1908–09	....	+	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....
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THE BLOOD-PRESSURE IN PNEUMONIA.*

ALEXANDER LAMBERT, M.D.

The treatment of pneumonia is still so empiric that any observations which may tend to assist in the treatment of this disease are worth reporting. A few observations on the blood-pressure in some forty-eight cases of pneumonia in adults are, for this reason, offered for consideration. The reports on the blood-pressure in pneumonia vary so greatly that there must have been heretofore great divergence in instruments used or an insufficient number of observations to enable any one to gather together into a connected whole the observations that have been made. Some observers class pneumonia as a disease with normal pressure, others as one of hypertension, and stilll others as one with abnormally low pressure. Giglioli reports some fifty cases measured with the Riva-Rocci instrument, and states that in favorable cases the changes in pressure were slight. In severe case the fall at the height of the disease on the fourth or fifth day was down to 80 or 90 mm. of pressure. In fatal cases rapid fall of pressure with dilatation of the heart is recorded. Others have found that a slight hypertension occurred during the first day or so, that in serious cases there was the rapid fall, the practically normal run of pressure in favorable cases, and that in favorable cases the pressure slowly returned to normal in convalescence. A drop down to 90 mm. of mercury has been considered of serious import. Gibson of Edinburgh, in studying the relation of blood-pressure to pulse and respiration, has endeavored to make some rule that would be of assistance in the treatment of pneumonia, or at least could be used as a warning of a beginning lack of equilibrium in the cardiovascular circulation, or even heralding a collapse. He has concluded that a general rule, the pressure in pneumonia is a little below normal with considerable variations throughout the course of the disease. The sudden fall of blood-pressure at the crisis has not occurred in Gibson's experience; hence, though reported by other observers, it cannot be very common. A pressure that is appreciably below normal is invariably of evil omen, and any considerable fall bodes disaster—and he sums up his experience in the following well-expressed observation:

When the arterial pressure expressed in millimeters of mercury does not fall below the pulse-rate expressed in beats per minute, the fact may be taken as of excellent augury, while the converse is equally true. That is, when the pulse-rate per minute is higher than the pressure of the millimeters of mercury, the equilibrium of the circulation is seriously disturbed.

C. A. Gordon has confirmed the practical value of Gibson's rule.

The following observations in forty-eight cases of pneumonia have been taken with a Janeway apparatus in Bellevue Hospital, and there has been an endeavor to take a daily measuring of the systolic blood-pressure of each patient. This has not always been possible, but in the cases which are reported a sufficient number of observations have been taken to give a fair judgment of the condition of the pressure in the course of the disease. These cases include both bronchopneumonia and lobar pneumonia, there being eight cases of bronchopneumonia and forty of lobar pneumonia. The ages have ranged from 13 to 78 years. They have been taken as they came to the hospital, of all ages, both sexes, and all conditions of life such as are found in an active general hospital service. They include the mild and moderately severe as well as the fatal cases

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—those of patients who were total abstainers from the use of alcohol, who used it in moderation, and who used it to excess. Of the eleven fatal cases which occurred, ten were among those who used alcohol, the other patient being a total abstainer. In such a small number of cases as here reported one hesitates to generalize, and one's observations must be recorded as facts and occurrences which may or may not be further corroborated. In the mild cases, in which there did not seem to be much toxemia, the disease did not seem to change the blood-pressure from the average normal in young adults, 120 to 130; it remained normal throughout their attack. In some young adults whose pulse remained below 120 and the blood-pressure remained above 105 during the disease, while there was no change at the crisis in the pressure, it fell below 100 after convalescence had set in, the pressure really being lower in convalescence than during the disease. These patients all recovered. In others the pulse remained below 120 and the blood-pressure below 105, and as soon as the crisis occurred and convalescence set in the pressure quickly and steadily rose to 120 to 130 mm. In no case did we see a sudden fall of pressure at the crisis. In some of the severe cases the blood-pressure has been low, the pulse, temperature and respiration very high, and this ratio has remained persistently throughout the disease. These are the cases in which Gibson's rule is the most noticeable and the patients are very ill and recover or die as the case may be. In those patients who recovered the pulse usually diminshed in frequency before the blood-pressure rose. In some few instances the reverse occurred. In other patients neither the temperature nor the pulse ran very high, and the blood-pressure was very low, below 100: still the ratio of Gibson's rule did not point to a bad prognosis, that is, the pulse-rate remained below the millimeter pressure of mercury. These patients recovered and the pressure persisted very low in their slow convalescence. In some patients suffering from chronic nephritis, the blood-pressure remained above 150 mm. but was distinctly lowered by the pneumonia. Some of these patients recovered and some died. In those who recovered the blood-pressure rose after the crisis. In other patients with nephritis, the blood-pressure has remained high (200 to 180) and yet the patients have died. In the cases of elderly patients with arteriosclerosis and in some cases of chronic nephritis, Gibson's rule did not hold good, and yet many of these patients died. The readings of the blood-pressure gave no hint of the cardiac failure. Taking the forty-eight patients, it is interesting to note that twenty of the forty-eight showed Gibson's rule, and of these six died. Of the twenty-eight that did not show Gibson's rule five died. As expressed in percentages, this shows that of those who showed Gibson's rule, 42.8 per cent. died, but of those who did not show it, 17.8 per cent. died. It is interesting to note among those who showed Gibson's rule that not quite half were alcoholics, and among those who did not show it not quite three-fourths were addicted to alcohol; but then among those who did not show it are included the arteriosclerotics and the nephritics, which further disturbs the ratio of blood-pressure and pulse-rate.

In summing up the subject Janeway remarks:

When one pictures the possible causes for variation in lobar pneumonia, the difference in individual reaction to the toxemia and in extent of lung tissue involved, the motor restlessness of some patients, the urgent dyspnea of others, and the great likelihood of an asphyxial rise of pressure where cyanosis is extreme, there is little wonder at these somewhat discordant results.

Looking at the figures alone in print without the patients, the results do seem extremely variable and discordant, but from impressions formed at the bed-side of watching the individual patient, of judging carefully the wisdom of

this or that order for medication, and using the knowledge obtained from the blood-pressure as well as from the pulse, temperature and respiration and general appearance of the patient, the impressions left are not so discordant as the mere grouping of types would seem. In the patients who were non-alcoholic, even among those who had passed the resistant stage of youth, Gibson's rule seems to hold particularly true. With an undisturbed blood-pressure, and with a pulse-rate well below the millimeters of mercury, one may feel easy in mind concerning the outlook, but where the pulse-rate runs higher than the millimeters of mercury of blood-pressure, there is a very distinct warning and danger-sign that cannot be disregarded. After convalescence has set in, the pulse-rate, however low the blood-pressure may be, usually remains well below the millimeters of mercury in number. Patients who have arteriosclerosis, and who have chronic nephritis, are not apt to show Gibson's rule, but the danger of cardiac failure is none the less very great. In the eleven fatal cases here recorded, five showed Gibson's rule and six did not. One cannot, therefore, attribute the break-down of the cardio-vascular equilibrium to one cause only, as many are apt to do. We hear much of vasomotor paralysis in pneumonia and infectious disease, and the figures here presented undoubtedly show that it is common enough in pneumonia, but it is not the only cause of fatal termination. The pressure may remain up, and there may be no vasomotor paralysis until a few moments before death, and the cause of death be due to a failure of the intrinsic muscles of the heart. I have often been struck, at the necropsy of patients who had died from what seemed to me a disproportionately small amount of infection in pneumonia, with the frequency in such cases of the brown friable heart muscle which seems to be summed up pathologically as brown atrophy. The great proportion of degenerated heart muscles in alcoholics has as much influence in the increased fatality of pneumonia in these patients as vasomotor paralysis. Of the eleven fatal cases here reported, one-half showed vasomotor paralysis, and half did not. The measurement of blood-pressure, therefore, will decide often the question of whether we should look to the heart or the blood-vessels to reestablish the equilibrium of the circulation. One hears recently a great deal about the use of epinephrin in cardiac failure in pneumonia, but unless one knows whether it is a vasomotor paralysis with a low-tension pulse, or a cardiac failure with a high-tension pulse, one certainly cannot use it intelligently, for while it is wise to give it with a low tension and it undoubtedly has done good in many cases in which this has been the factor against which the epinephrin was working, it is unquestionably poor judgment to use it with a patient whose blood-pressure is already high, and whose heart is struggling without sufficient reserve force against too great a strain. On the other hand, one sees a mixture of strychnine, camphor and alcohol given to raise a falling blood-pressure. One cannot acquiesce in the wisdom of this, for therapeutically it is a neutral mixture. The alcohol will dilate the vessels and reduce the blood-pressure vastly more than the strychnine and camphor can raise it. I have heard it reported that such a mixture had been given all day to raise the blood-pressure, and the situation saved by epinephrin. One always wonders if the epinephrin would have been necessary if the alcohol had been left out. It is also a grave question whether alcohol is advisable in pneumonia until we know whether the blood-pressure is high or low, for if it is lower than normal, we certainly should not desire to further reduce it by such an effective means of lowering the blood-pressure and dilating the vessels as is alcohol. There certainly are patients with whom we are required to use all the means in our power, as camphor, strychnine, digitalis and epinephrin, to prevent a vasomotor collapse, and there are other patients with whom we must

equally endeavor with vasomotor dilators to reduce the tension in the pulse to the amount against which an overstrained heart can best work and carry on its share in the equilibrium of the circulation. The blood-pressure instruments to-day are of such convenience that they offer the easy opportunity to ascertain at a visit these facts. A knowledge of the blood-pressure in pneumonia will often be of assistance in judging of prognosis and of treatment. These are the two vital points to the patient.

THE TREATMENT OF ACUTE LOBAR PNEUMONIA.*

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From time to time an article will appear in current medical literature advocating some special line of treatment for pneumonia, and claiming a remarkedly low death rate for the cases so treated. Such articles are usually based on a limited number of cases, occurring in the same locality and in the practice of one individual, whose temporary success in a series of cases awakens his enthusiasm. But when such statistics are compared with those of a vast number of cases collected from the general hospital and private practice throughout the United States, as was done by Wells in 1904, (1) it will be seen that pneumonia still continues to exact its toll of 20 to 25 per cent. of deaths. Thus, of 465,400 cases, Wells found a mortality of 20.4 per cent., and among 43,455 cases collected by Norris and Musser 21.06 per cent. died. (2) These figures form a striking contrast to the reports of some observers, who claim a percentage recovery of from 95 to 100 per cent. in a limited number of cases treated according to some pet plan.

It is of course understood that pneumonia is an acute generalized infectious septicemia, with a localized expression in the lung; yet many practitioners seem to be unable to rid themselves of the obsession that the lung must furnish the sole object of investigation and attack. We read of measures addressed to the local condition as if this alone were the enemy to be overcome; yet the lung presents exactly the same condition after the crisis, when the danger is over, as it did a few hours before, when the patient's life trembled in the balance. Patients may be overwhelmed by the general toxemia when the physical signs show little or no lung involvement, or may exhibit extensive lung involvement, with little or no general disturbance.

The patient with the pneumonia, and not the pneumonia that is with this particular patient, must furnish then our fundamental concept of the management of the individual case. The obvious corollary is that there can be no specific adapted to every case, but that, inasmuch as the disease is self-limited and terminates by crisis, our efforts must be directed to facilitating the crisis, supporting the patient, preserving him from unnecessary stress and strain, and meeting emergencies as they may arise. Clinically, we meet with three classes of cases: (1) those who will recover in spite of all you may do to them; (2) those who will die in spite of all you can do for them; (3) a very large intermediate class in which the result may depend upon skillful, judicious therapeutic intervention. During an epidemic of pneumonia, or during the prevalence of influenza, which is so often complicated by pneumonia, certain measures of prophylaxis are advisable for the individual, for the community, and for the

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physician himself. Individual prophylaxis is best secured by keeping the nose, mouth, and throat as clean as possible by antiseptic sprays, mouth washes, and gargles; by the avoidance of crowds of coughing and sneezing persons; by abstaining from the use of alcohol, which paralyzes the sentient sentinel nerves of the respiratory mucous membrane, and by guarding against exposure, exhaustion, and privation.

Communal protection demands that the infected person should be isolated and his discharges carefully disinfected. The physician who takes charge of a case of pneumonia assumes a grave personal risk, and should have thorough knowledge of the disease, sufficient physical endurance, freedom from alcoholic or drug habits, unclouded judgment, executive ability, and self-confidence (3). Even then pneumonia cannot be prevented, but it need not be invited. A number of our modest brethren allow their patients to believe that a "threatened pneumonia" can be aborted, but it is hardly necessary to say to this audience that any abortive treatment of acute lobar pneumonia is theoretically impossible.

Given a case of pneumonia then, in private practice, the first question that naturally arises is, have we a specific for this generalized septicemia? If all pneumonias were caused by the pneumococcus, and if all pneumococci were alike, we might hope for an affirmative answer. At present the answer is—no. Our dependence then must be on the general measures to meet the indications previously outlined, and I have nothing new to offer you on that heading, but only a recital of the means that have helped me in hospital and private practice during thirty years. As soon after the initial chill as possible, the patient is put into a warm bed and given a mustard foot bath in the bed, under extra covering and with vigorous rubbing of the legs for thirty minutes. He is then given an enema of two ounces of epsom salts with two ounces of glycerin and twelve ounces of water. I avoid the use of the customary hypodermic of morphine at this stage unless pain is so great as to be uncontrollable by strapping the chest and by hot applications, or unless nervous excitement is so great as to seriously disturb circulatory equilibrium. My next order is for either ten grains of blue mass or a powder of calomel, five grains, with sodium bicarbonate, fifteen grains, to be followed in six hours by a saline laxative. Thereafter and during the progress of the disease I order a high, hot, large normal salt solution irrigation every morning, and also the epsom salts and glycerin enema every evening, because I believe in stimulating the splanchnic circulation, keeping the lower bowel empty, and preventing the development of troublesome tympanites later in the disease. For the same reasons I avoid overfeeding the patients, avoid articles of diet which may produce flatulence, and forbid the use of beverages containing carbonic acid gas. Patients are fed, however, every three hours with milk, eggs, broths, coffee, and other fluid foods, with water enough to make up an aggregate of five pints in twenty-four hours. They are not awakened from sleep at night. Sleep should not only be encouraged, but secured if need be by one of the newer hypnotics, or by morphine if you must, before it passes into marked insomnia with exaltation or delirium. A delirious patient requires constant vigilance. The sick room should be well lighted, and solicitous ignorance on the part of relations should not stand in the way of the unlimited admission of free, fresh, flowing air. It seems to me, however, that we have been carried away by the glowing accounts of success in pneumonia by putting patients out on balconies and roofs. Pure air at a temperature of 65 degrees F. has always satisfied me, with the exclusion of visitors and of the consideration of any business matters. The nurse should be efficient but not meddlesome. Too much meddlesome medication does more harm than good. Drugs and food should be given together if possible, and the patient's toilet wants should be attended

to at three-hour intervals, and he should have absolute quietude of mind and body between these intervals.

Next I endeavor to have an accurate blood count made, including a differential enumeration of the leucocytes, and to have a thorough uranalysis done. A daily blood chart thereafter, with a daily examination of a twenty-four hours' specimen of urine, will keep us alert for complications. A comparison of the blood pressure with the pulse rate will also give us valuable prognostic and therapeutic indications. If the pulse rate is less than the blood-pressure reading the patient is doing very well. Some clinicians believe that the remarkable reduction of arterial pressure which accompanies the onset of the disease should be encouraged, and such practitioners administer remedies to favor this circulatory depression, such as veratrum viride, aconite, nitro-glycerin, iodide of potassium, etc. Others presume that Nature is mistaken, and endeavor to correct her error with digitalis, adrenalin, etc. I am one of those who believe that low blood pressure can only result in vasomotor paralysis with consequent stagnation of the bloodstream, so that the tissue cells lose their accustomed stimulus and are consequently constantly bathed in a solution of their own wastes (3). Therefore I use from the outset strychnine, caffeine, alcohol, and camphor. I have not had the same success with ergot that has been claimed by some of my colleagues. Nor do I place much reliance upon digitalis in the presence of high fever. After the crisis, however, digitalis may be clearly indicated. Adrenalin is a drug which, when given hypodermatically in good-sized doses, helps in tiding over the crisis.

The fever in pneumonia causes the relatives and sometimes the attending physician, a great deal of unnecessary anxiety. Fever is a specific reaction against injurious materials which affect the tissues, and is, in its essentials, a protective reaction (4). Accepting this definition, fever requires no interference unless it becomes high enough to add to the poisoning of the vital centers. A temperature of 104 degrees F., associated with a moderate leucocyte count, with a blood pressure which is higher in the manometer reading than the pulse rate, and with clear heart sounds, gives a better outlook than does a temperature of 102 degrees F., with a low leucocyte reaction, with low systolic arterial pressure, and a muffled first sound with a pulse of 120. Hyperpyrexia, however, indicates danger, and should be promptly met by the application of cold compresses, cold spongings, and ice bags to the chest. Coal tar antipyretics should never be used. Quinine still has some ardent advocates, and its hypodermatic use has lately been extolled for its chemical and anti-toxic, rather than its antipyretic, effect (5). But, inasmuch as it is the death of the pneumococci, and not the neutralization of the toxins, which must precede the recovery of the patient, this action of quinine would seem to be inadequate. Personally, I believe that alcohol is a good antipyretic. Alcohol reduces temperature by increasing heat loss by evaporation and radiation; it lessens heat production; and, most valuable of all, it supplies an easily oxidizable fuel to be burned up instead of the tissues. In patients accustomed to its use, especially in the alcoholic pneumonias so numerous in Bellevue Hospital, I use it with a free hand, in half-ounce doses frequently repeated, unless coexisting kidney complications contraindicate. Its drawback is that it promotes vascular relaxation, but this risk must be accepted when the alcohol is needed.

The pleuritic pain of the early stage of pneumonia is usually severe, sometimes agonizing. If possible, I avoid the use of morphine, which obscures the symptomatic field, dulls the nervous reflexes, and causes an insistent demand for its repetition by the patient, which is dangerous to accede to. Strapping the chest, local applications, the Paquelin cautery, and mental encouragement should

be used to the limit, before morphine. Cough may also be distressing in the early stages, but here again the use of any opium preparation is a two-edged sword.

After the stage of onset, the toxemia of pneumonia becomes manifest in the functions of the cerebrum of the cardiovascular and of the respiratory systems. The first brain symptoms are excitation, restlessness, insomnia, and delirium, followed in severe cases by depression, stupor, and coma. The measures already described, of aerotherapy, hydrotherapy, and elimination by the bowels, skin, and kidneys, have done something to forestall these dangers, but if pronounced they may require enteroclyses of physiological salt solution, or the Murphy drop method, with increase of diaphoresis by hot packs, and an ice helmet to control delirium. Insomnia may require trional in hot milk, chloralamid in cold whiskey, or, if unavoidable, ⅛ grain morphine hypodermatically. The cardiovascular symptoms are harder to conquer, and, when pronounced, compel us to use our remedies by intramuscular, rather than hypodermic, injection, since Meltzer has shown that muscular tissue contains such a network of veins that injections therein are almost as promptly taken up as when given intravenously. My own faith rests firmly on strychnine and alcohol for steady and continuous effects, despite the doubting Thomases of the pharmacological laboratories. I hold camphor in sterilized oil and adrenalin in reserve for emergencies, and believe that I have seen good results in Bellevue from the intravenous injection of 1 milligram of strophanthin in cardiac collapse, after the failure of the first mentioned remedies.

The dyspnea of mechanical obstruction by involvement of large portions of lungs is best relieved by oxygen. Not the canned oxygen supplied by manufacturers, but that of the free, fresh, flowing air. Edema of the lungs, if dependent upon low arterial pressure, will be helped by intramascular injection of adrenalin, which raises the blood pressure by stimulating the vasoconstrictor fibers of the splanchnic vessels and thus drives the blood into the brain, lungs, and heart. If associated with high blood pressure, artificial respiration seems to facilitate the discharge of serum from the tubes and to facilitate the pulmonary circulation (6).

When your patient has safely come through all these dangers, and has entered upon convalescence, keep him in bed a week longer, carefully estimating each day the working power of his heart, and keep him a second week in a chair on a sunny porch. The complications of pneumonia are many and cannot be touched upon within the time limits of this paper. One word only. Cases of "unresolved pneumonia" will resolve themselves into post-pneumoniac empyema upon the sterile introduction of an aspirating needle.

If I may be allowed to summarize this discursive recital of an old story, I may say that the factors upon which I hope for success in the treatment of pneumonia are isolation, ventilation, disinfection, elimination, and support. The factors making for fatality are half enough air, half enough water, half enough rest, too much meddlesome medication. Our search for a specific has hitherto been fruitless. Serum therapy, from which so much was hoped, has thus far signally failed. There are several reasons for this failure which, however, need not be gone over here.

In our line of treatment we must consider two things: the action of the infecting organism on the tissues: the defensive powers of the host. With the former we can do very little by therapeutic means. The latter must be fortified by every means at our command. Death ensues from an overwhelming extent of the inflammation, or from toxemia, producing the condition known as cardiac failure. The entering wedge of this condition is laid at the very beginning of the

attack in the vasomotor paresis which transfers a great excess of blood from the arterial to the venous circulation, and this loss of circulatory equilibrium must be fought from the very outset. The time to treat "heart failure" is before it has developed. The physician who withholds cardiac support until his patient presents a dry brown and fissured tongue, low muttering delirium, a pulse rate of over 120 with a low arterial pressure, and a feeble first mitral sound with a failing second pulmonic sound has lost his golden opportunity. In spite of all our measures of support, this condition may develop, and it is here that venesection sometimes gives brilliant results by abstracting a pint of toxin-laden blood, and diluting the remainder by introducing liquids by the mouth or rectum, or by hypodermoclysis. Venesection should never be done in the very young, the very old, the anemic, or the weak. It is not indicated in the stage of onset, as the formation of the exudate at that time consumes from 2 to 4 pints of blood.

The view that heart failure is due to the poverty of calcium in the circulating blood in pneumonia has recently been put forward, and the administration of calcium chloride in 10-grain doses every three hours is eloquently advocated (7). For the same reason, the free use of sodium chloride has been urged. These remedies have no deleterious effects, and their use may be added to that of the other measures herein recommended.

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ADRENALIN CHLORIDE IN THE TREATMENT OF CARDIO-VASCULAR COMPLICATIONS OF LOBAR PNEUMONIA.*

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I have presumed to consider this subject not because it is a new one, but because, as a result of the study and observation of cases of lobar pneumonia and especially those accompanied by cardio-vascular failure and pulmonary edema in which we have used adrenalin chloride, I have been impressed with the belief that we can establish a definite clinical picture the presence of which will immediately suggest the administration of adrenalin chloride, while the absence of this picture will be as quickly recognized as a contra-indication for this plan of treatment.

The physiological action of this drug is definite and yet so pronounced that favorable results can only be obtained by using it in carefully selected cases: otherwise very unfortunate results will follow its indiscriminate use, especially in cardio-vascular failure and pulmonary edema accompanying lobar pneumonia. In considering this subject we have excluded all complications and causes of death other than those directly related to the cardio-vascular system. Before considering the administration of adrenalin, I believe that we should separate our cases of cardio-vascular failure with pulmonary edema or impending edema, into three groups, each group having a distinct clinical picture.

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Group I.—Cardio-vascular failure and pulmonary edema resulting from toxic paralysis of the centres of the heart. This group is small in number and usually occurs in the course of pneumonia suddenly and without warning, resulting in death in a short time, the heart refusing to respond to any form of treatment or stimulation. These cases do not present any definite premonitory symptoms either of acute dilatation or loss of control of the peripheral vessels.

Group II.—Acute dilatation of the heart due to toxic degeneration of the muscle wall or added to the myocardial degeneration of people in advanced years. This condition is accompanied by an elevation of pressure usually ranging from 125 to 170 mm. mercury. Cyanosis is a marked symptom from the beginning in these cases. The superficial vessels are contracted and the hands and feet are cold. The first sound of the heart loses its muscular quality. The pulse is rather small, of high tension, irregular both in frequency and size, the irregularity in size being very important as showing an incomplete systole, and this seldom occurs in acute diseases unless indicating a serious overtiring of the heart. These symptoms are present and more or less prominent until relieved by crisis, but they may be replaced at any time by a sudden attack of dyspnea, increase in the cyanosis, a pulse increasing in rate but retaining its previous characteristics. Physical examination will reveal definite signs of enlargement of the heart, with descent of the apex, etc. Adrenalin if used in these cases will intensify the dilatation by raising the pressure, as illustrated in the following case:

Group II.—Case I. F. B., admitted to the hospital delirious and not in a condition to obtain history. Right lower lobe involved. Leucocyte count 19,000. At the time of admission, pulse 100 to 120, respiration 36. Blood pressure on the right 150, 140 on the left. Cyanosis marked. Pulse small, high tension, dyspnea extreme. Patient presented all the contrasting symptoms as compared with Group 3. Death due to cardiac dilatation accompanied by pulmonary edema.

Group III.—Vaso-motor paresis. In this group we have a condition resulting from toxemia which affects excessively the control of the calibre of the arteries. The average well-balanced pneumonia with a temperature ranging between 103 and 104, and with a blood pressure of 95 to 110, should be accompanied with a pulse ranging between 96 and 106. The pulse is soft and compressible, and on the third or fourth day may even be dicrotic. Cyanosis is not a prominent feature as in Group 2 and the extremities are warm. While the above symptoms are all favorable, the blood pressure should be carefully watched and a daily estimation made, as an impending loss of vascular control will be anticipated by a fall in the pressure and an increase in the rate of the pulse from 96 or 110 to 120 or 140. This tendency to increase may not be constant during all of the twenty-four hours, but will be a sufficiently prominent feature of the chart to be easily detected if followed closely, and will be confirmed by the compressible pulse and warm extremities. This low pressure and fast pulse may exist for several days without exciting any great anxiety and be entirely relieved by a favorable termination, but on the other hand, not infrequently about the fifth or sixth day there may be a sudden acceleration of the pulse, dyspnea, cyanosis, a drop in the blood pressure to 70 or 80 mm., pulmonary edema and death before the racing heart can be controlled. These symptoms have been observed not only during the activity of the disease, but also at the time of the crisis and during the decline of the temperature by lysis accompanying delayed resolution. The heart is rapid but does not display the signs of dilatation.

The following have been selected as illustrating the clinical features of this group:

Group III.—Case I. J. K., admitted to the hospital on the fourth day of pneumonia, left lower lobe. At the time of admission the blood pressure was 110, respiration between 28-30; the pulse showing a decided tendency to be disturbed in rhythm, ranging on the fourth day between 114 and 124. On the fifth day the pulse ranged between 112-128; sixth day, 114-138; seventh day, 120-138; eighth day, 118-140; ninth day, 130-142; so that the rapidity of the pulse, out of all proportion to the temperature, blood pressure and respiration, was strongly marked.

This patient on the 19th (or sixth day of his disease) developed signs of lack of compensation and stimulation was administered as follows; digalen. minims 10; whiskey ½ oz.; strychnine 1-40; every four hours. This was continued, and on the 22nd or ninth day, he developed a slight pulmonary edema and his blood pressure, when estimated, was found to be 70 on the right and 88 on the left. A series of adrenalin chloride was administered, the patient receiving 15 minims every twenty minutes for six doses.

It will be noted that this disturbance in the loss of control in the circulation and edema occurred at the time of crisis. The pressure, as a result of the injection, increased to 90 on the right and 105 on the left. The pulse dropped from a maximum of 142 on the 22nd to a maximum of 112 on the following day. As the pulse still displayed low tension qualities on the 23rd, the day following the edema, two additional doses of adrenalin were given and then discontinued.

During all this time, that is, from the 20th through the 23rd, the digalen, whiskey and strychnine had been continued, but without any evidence of a control of the heart. With the restoration of the vascular equilibrium, the tension began to rise, so that on the 25th a pressure of 150-160 was present, the pulse ranging between 94 and 102. It would seem that during the period of loss of vascular control, between the 19th and 23rd, it was impossible to obtain a vaso-motor constriction from the use of the digitalis.

Group III.—Case 2. J. M., admitted to the hospital delirious on the 16th and not in a condition to obtain history. The day after admission the blood pressure was 105, pulse showing a tendency to increase with a decreasing temperature. This feature is especially noticeable on the 17th, 18th and 19th. In spite of stimulation, the patient developed a pulmonary edema on the 19th, having at the time a pressure of 100 mm. mercury, and died on the 20th, although on the last day of his illness a slight slowing of the pulse was evident.

Group III.—Case 3. A. C., admitted on the 7th day of lobar pneumonia. An unfavorable prognosis was made in this case as a result of the observation of the low pressure with a tendency of the pulse to become rapid out of proportion to the temperature, physical signs and the absence of dilatation of the heart. Range of pulse 104-150, blood pressure 95-105.

Group III.—Case 4. R. G., admitted to the hospital on the 31st, with lobar pneumonia of right and left lower lobes. This patient throughout the course of the disease has been relatively comfortable, cyanosis scarcely noticeable blood pressure ranging between 80 and 116 maximum; pulse ranging between 100 and 114, the symptoms being well balanced and the patient's condition being entirely satisfactory. The blood pressure should be carefully observed daily, having in mind the possibility of a vasco-motor collapse. Should the patient show evidence of an increasing pulse out of proportion to the change in pressure,

small doses of adrenalin should be given to steady the pressure and thus prevent a complete collapse and subsequent edema.

The comparison of the clinical pictures of Case 2, Group II, and the cases classed in Group III was very striking. All patients were admitted to the ward with lobar pneumonia. In Case 2, Group II, (T. M.) at the time of admission the patient was cyanotic, dyspneic, pulse was small, high tension. Blood pressure on the right was 150, on the left 154. Heart was laboring and the patient presented all the symptoms necessary for an unqualified and poor prognosis. The blood pressure increased until it reached 180mm. on the fifth day of the disease. The case terminated on the sixth day unfavorably.

All of these cases except Case 2, Group II, were surprisingly free from cyanosis until the development of the terminal symptoms in the fatal cases.

This condition of vaso-motor dilatation or paresis is accompanied by a clinical picture which is sufficiently definite to make it possible to differentiate it from the cases classed in groups 1 and 2 and as the only type in which we should use adrenalin. For, if our deductions are correct, its use in Group I, would be useless and in Group II, detrimental and dangerous as illustrated by the following case:

J. K., admitted to third division with pneumonia, right lower lobe. Temperature 101½, respiration 32, blood pressure 120 mm. pulse rate proportionate. On the seventh and eighth days he was given adrenalin experimentally, 15 minims every three hours. The pulse at the time ranged between 106 and 116, and the respiration 32-42. On the 9th day the temperature dropped rapidly from 103.4 to 100, the pulse increased to 128 and his dyspnea and cyanosis increased, his pressure at this time being 185 mm. The adrenalin was discontinued at 8 a.m. and at 4.45 the blood pressure had dropped to 135 mm., the pulse to 108, and from this point the patient continued to improve. The continuation of the adrenalin would, I believe, have induced dilatation of the heart.

The cases in Group III may best be compared to a steam engine running under a normal pressure of steam which is subjected to the sudden withdrawal of the load or resistance. The immediate result of this will be a racing and destruction of the engine, unless the load or resistance can be quickly reintroduced. A similar condition exists in the heart under these circumstances and as the vessels fail to respond to the vaso-motor control, the heart will increase in rate and be accompanied by pulmonary edema, death intervening due to an exhausted heart. The problem of treatment is therefore to introduce some substance which will quickly cause a contraction of the peripheral vessels and act as a governor. It is necessary that this peripheral resistance should be sustained for some time to be of any great value. To determine whether adrenalin possessed this property, we conducted a series of experimental injections, selecting the inter-muscular administration in preference to administration by mouth, because of the uncertainty of action when used in this latter way, and in preference to the intravenous method, because of its slow absorption from the muscle. For these experiments we selected patients between the ages of 20 to 50 whose arteries should still retain the possibilities of contraction and dilatation. All of these patients were given 15 minims every twenty minutes for four doses, the pressure being taken every hour thereafter until it returned to the low point. We found the pressure would be maintained above the point recorded before the injection for about four hours and the rise would be noticeable about fifteen minutes after the first injection, the maximum pressure being reached in about one and three quarters to two hours from the time of the first injection.

A second series was given to some of these patients resulting in a second

rise sustained for about the same length of time, thereby demonstrating that the blood pressure would be maintained for a considerable period and a perfect response to a second series if it became necessary to maintain the pressure for a longer period of time.

In conclusion, therefore, I would advise the inter-muscular injection of adrenalin chloride only in cases of impending pulmonary edema if they present the clinical picture described in Group III.

That it should be used in 10 minim doses before any signs of edema appear provided the pulse shows a marked tendency to increase in rate and the blood pressure is below 110 and the other symptoms of vaso-motor dilatation are present. If edema develops suddenly during a pneumonia that it be used in 15 minim doses every twenty minutes for a series of four to six doses or until the symptoms are controlled and that this series be repeated if there are signs from the edema accompanying acute dilatation of the heart, by applying symptoms occurring in Group 3.

STUDIES OF THE LEUKOCYTES IN PULMONARY TUBERCULOSIS AND PNEUMONIA.*

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It has long been recognized that the leukocytes of the circulating blood play an important role in many, if not all, bacterial infections, and numerous investigations have already contributed valuable additions to our knowledge of this subject.

The researches of Arneth, calling attention to the morphology of the nucleus in the neutrophil leukocytes, especially in tuberculosis, have stimulated further studies in this direction on the part of various observers. The results reported in this paper were first suggested by the work of Arneth and represent an effort to corroborate and amplify his results by various experimental and clinical observations and especially to determine their value in the diagnosis and prognosis of pulmonary tuberculosis and pneumonia.

It seemed possible that Arneth had laid undue emphasis on the significance of the neutrophil leukocytes and that it was important to study the other forms of leukocytes in connection with them. Pneumonia was chosen as a companion study with pulmonary tuberculosis, as forming a contrast of an acute with a chronic pulmonary disease.

An effort has been made to collect the information which is widely distributed in the literature, and by correlating our own results with that of others, to try to present a fairly complete study of the behavior of the white blood-cells in these two diseases.

The clinical material was obtained from the tuberculosis clinic, day camp and wards of Bellevue Hospital, and in the case of pneumonia from the medical wards of Bellevue Hospital, many of the cases observed being under the services of Dr. George R. Lockwood and Dr. William K. Draper, to both of whom we are deeply indebted for the privilege of studying these cases.

The Leukocytes in Normal Individuals.

As a basis of such a study, it is essential to establish the normal conditions both as to total number of leukocytes in the blood and as to their distribution among the various classes as shown by the differential count.

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Almost all authorities place the normal number from 5,000 to 10,000 per cubic millimeter. The text-books have evidently appropriated these results from the earlier observations of Hayem (1), Türk (2), Grawitz (3), Naegeli (4) and others. A recent careful study by Kjer-Petersen (5), however, places the average normal number between 4,000 and 5,000 in men and comes to the conclusion that in women the normal count is very irregular, varying from 4,000 to 25,000 under various physiological conditions.

This observer lays great stress on the fact that the blood is often not homogenous, this being especially apt to be the case in women. He attributes the higher counts usually noted to improper methods, laying particular stress on the necessity of taking the specimen of blood when the subject is fasting before breakfast and also on the importance of invariable close approximation of the cover-slip to the counting chamber so as to produce Newton's rings. Arneth, Steffen, and others agree with Kjer-Petersen. In our own studies we found the average number of leukocytes in a large number of counts to be 9,000.

In the normal differential count there is considerable variation among the various authorities. This is largely due, however, to the fact that there is no unanimity of opinion as to what types of cells are included in the various groups. Table 1 gives the figures quoted by several independent observers.

Table 1.—Normal Differential Count.

	Poly-nuclears.	Trans-itionals.	Large Mono-nuclears.	Large Lympho-cytes.	Small Lympho-cytes.	Eosino-phils.	Baso-phils.
Cabot (6)	62–70	..	..	4–8	20–30	0.5–4	1/45–0.9
Ewing (7)	70–72	2	4	22	25	2–4	0.5–2
Wood (8)	65–75	2–4	1	22	25	2–4	0.5
Naegeli (4)	65–70	3	5	22	25	2–4	0.5
Webb and Williams (9).	54	..	..	24	10	2	0.4
Da Costa (10)	60–75	4	8	..	20–30	0.5–5	0.5

Many emphasize the importance of calculating the actual number of each class of cells rather than indicating the results by percentages. In our work we have discarded the terms "transitional" and "large mononuclear," and in our differential count these cells, as described by others are probably partly included in the polynuclears and partly in the large lymphocytes.

The Biology of the Leukoycytes.

In order to understand properly the significance of the differential count of the leukocytes, particularly the work of Arneth (11), it is valuable to summarize briefly the theories concerning the development of the leukocyte.

There are two general theories regarding this development, dividing hematologists into two distinct schools. One, under the leadership of Ehrlich (12), considers that the polynuclear neutrophils, eosinophils and basophils are derived only from their corresponding marrow-cells and that there is never a transition from the lymphocytes to the polynuclear forms. The lymphocyte, according to this school, develops from the various lymphoid organs, some even from lymphoid tissue in the marrow and pass through the various stages of lymphoblasts, large lymphocytes and small lymphocytes, but never into the types of cells with granular cell bodies.

The large mononuclears and transitional cells, according to this school, are separate entities derived from the marrow and the transitional cells probably are derived from large mononuclears and in turn are transformed into neutrophils. Pappenheimer (13), Naegeli (4), Weidenreich (14) and the majority of observers now accept this theory.

The other theory, which is held by the Russian school under the leadership of Uskow, and which is also supported by Grawitz, holds that the neutrophil cells are frequently derived from the lymphocytes, and that the so-called transitionals often represent a stage between these two types of cells.

Practically all authorities agree that the true lymphocytes do not act as phagocytes, although it has been demonstrated that they do in some way hinder the development of the bacterial infection in the body.

In the development of the neutrophils, Arneth's contention is that their progress from the myelocyte is marked by changes in the nucleus, it being first indented and bent and finally lobulated, the mononuclear forms, therefore, representing the younger and the multimononuclear forms the older type of cells. The granules also are more numerous in the older cells. In the younger cells the granules are apt to be first basophilic and later oxyphilic. The origin and significance of the eosinophils have been carefully studied by Brown, but accurate scientific knowledge of them is still lacking.

Arneth's Blood-Picture.

In developing his theory of the changes of the nucleus during the growth of the neutrophil, Arneth has divided the neutrophils into various classes according to the number and shape of the nuclear segments. This he tabulates into a very involved blood-picture. His contention, supported by numerous clinical observations, is that in various diseases the differential count of the neutrophils, according to his scheme, very often shows an abnormal predominance of the younger forms of cells, which he calls a shifting to the left in his blood-picture, that this shifting is very characteristic and represents a definite reaction on the part of the organism to the infection, and that a study of these changes forms a valuable basis for the differentiation of diseases and knowledge of their clinical course and subsequent history.

Arneth's work has attracted a good deal of attention and many reports based on his theory have appeared in the literature. Hiller (15), in addition to finding Arneth's method unreliable clinically because of the great variations in the normal, and because of the fact that the changes noted bore no relation to the severity of the disease, disagrees with the theory of Arneth, contending that the neutrophils are derived from lymphocytes. Pappenheim (13) successfully refutes his contentions. Pollitzer (16) dissents very strongly from Arneth as to his theory of development of the nucleus, and also holds that the changes in the nucleus noted are due to artefacts caused by the method of staining.

Arneth's reply to our own investigations discredits Pollitzer's contentions. Busse (17) objects on the ground that experiments showed that there was no variation of phagocytic power among the different classes of neutrophils according to the number of nuclear segments. He consequently argues against Arneth's conclusions that these variations in the nucleus denote variations in efficiency against infection. Arneth, in his reply, denies the importance of these observations.

Zangemeister and Ganz (18) object to Arneth's method of considering the neutrophils to the exclusion of the other forms of leukocytes and also to Arneth's classification. Their experiments with streptococci tend to show that the mononuclear neutrophils are the most important from the standpoint of prognosis, the polynuclear neutrophils and the eosinophils coming next in order.

Flesch and Schossberger (19) studied many diseases in children, and while agreeing to the fact that the shifting to the left did occur under various conditions, found that this shift had no relation to the clinical course of the disease. Pollicek (20) also found that in tuberculosis the blood-picture was of no value

clinically either in diagnosis or prognosis. Bourmoff and Brugsch (21) came to similar conclusions. In this country Kagan (22) and Solis Cohen and Strickler (23) found Arneth's method of no clinical value.

Many other observers, however, particularly recently, have corroborated Arneth. Among these are Bochenski (24), Kownatski (25) and Grafenberg (26) in sepsis, particularly in puerperal fever; Kohl (27) and Kothe (28) in appendicitis, Connenberg and Kothe (29) in peritonitis, Esser (30) in intestinal diseases of children and Lewinson in several diseases, found Arneth's method of value both in diagnosis and prognosis. Many others have corroborated Arneth's methods in tuberculosis. These will be noted more in detail later.

Blood-Picture Indices.

Many observers, including ourselves, have found Arneth's method very cumbersome on account of the multiplicity of detail and on account of its consequent lack of definiteness, which impairs its clinical value. Various suggestions have been made by which the variations in the nucleus of the neutrophils might be noted by a definite index. For this purpose some use as an index simply the number of the neutrophils which are mononuclear, claiming this as a sufficient measure of the shifting to the left. These observers are Zangemeister and Ganz (18), Kothe (28), Sonnenberg and Kothe (29) and Kohl (27), Others, including Klebs (31), Dluski and Rospedzihowski (32) and Minor and Ringer (33), use the sum of Classes 1 and 2 of Arneth's picture as an index.

Adolph Wolf (34) advocates a total nuclear count of the neutrophils, counting the round segments as one and the bent or loop forms as one and one-half, and in counting he notes only the number of these two types of nuclei and totals them for his nuclear counts. Von Bonsdorff (35) has a similar method somewhat modified. Sabrazès (36) has worked out a neutroleukocytic and neutro-nucleo-leukocytic quotient as an index. Bushnell and Treuholtz (37) use the sum of Classes 1 and 2 and one-half of Class 3 as an index. In our observations we have followed this suggestion, as it seems to lose less of the value of Arneth's original classification than do the others.

Technic.

In our observations the specimens of blood are usually, but not invariably, obtained in the afternoon from three to four hours after the ingestion of food. The other more usual causes of physiological leukocytosis, such as exercise and cold baths, were avoided. Also, no cases were studied after hemorrhage.

We emphasize, as have others, the importance of obtaining thin and even smears of the blood for differential count and of close approximation of the cover slips to the counting chamber for enumeration.

After numerous experiments in various methods of fixing and staining, we found that fixing by drying in the air and passing the slide two or three times across the flame, and the use of Wright's stain made up fresh produced the best results for the study of the nuclei. Enough stain is dropped on the slide to cover the smear, and the dish then covered for one minute. It is most important to have fresh stain often, as Wright's stain does not keep well.

The slide is left for two or three minutes with the stain, at the end of which time eight or ten drops of tap water are dropped on and the dish again covered for one minute or until the metallic scum appears, when the slide is washed by dipping into a glass of distilled water and out quickly, when it is blotted and dried. All counts should be made with the oil immersion lens.

One hundred neutrophil cells were counted in each observation and the other forms of cells found during this count were noted, so that an average of 140 to 150 leukocytes were counted in each observation.

Many slides were studied by two or three separate observers, and the similarity of the results in the Arneth counting satisfied us that it is an accurate clinical procedure.

In differentiating segments of the nuclei, superimposed segments were always counted separately, as also were those connected by thread. Those having a connecting isthmus, however, were counted as one segment.

Experimental Work.

Many experiments were carried out to aid in as complete an understanding of the changes found in the white blood-cells as possible, especially in relation to Arneth's studies with the neutrophils.

These experiments can be summarized under the following headings:

1. **Staining Experiments:** These were undertaken to determine which stain would give the best nuclear picture, and at the same time to differentiate the different classes of leukocytes in the blood.

Smears were prepared in the usual manner except that the slides were clean and as sterile as possible and the blood was spread in a thin and even film. The smears were killed in all the well-known cytological methods and each method of killing and fixing was stained by the three usual differential blood-stains, that is, Wright's differential, Jenner's differential and the eosin and methyl blue method. Many other slides were stained with iron hematoxylin, Ehrlich's triacid stain, and with Goldsmith's triple stain.

The results from these experiments showed that the best method for preparing the slides was to kill the blood-smear by simple drying and to fix and stain with Wright's differential stain, which gave the best nuclear picture as well as differentiated the leukocytes. Jenner's stain does not give a good nuclear stain and fades rapidly, so it was not used.

For the cytological study of the nucleus, slides killed with hot corrosive sublimate and stained with iron hematoxylin gave the best picture. The usual method for the study of the nucleus, i. e., to kill with Flemming strong and stain with iron hematoxylin did not give satisfactory results with the blood smears.

2. **The Question of Artefacts:** These experiments were suggested by Pollitzer's article (16), in which an attempt is made to prove that the number of pieces of lobes found in the nucleus of the neutrophils is an artefact and has no connection with the state of the nucleus in the living neutrophil.

(a) Smears were prepared from the blood of three subjects, each of which showed an Arneth index slightly different from the other. These smears were prepared in all the known cytological methods and numbered with a key so that at the time they were counted it was not known from which subject the slide had been prepared. It was found that the smear from each subject gave practically the same index, even where the preparation had been so poor as to be almost useless and that the difference between the indices of the blood-pictures from the three subjects remained the same as in the smears prepared by Wright's differential method.

(b) Smears were prepared in which an attempt was made to stretch and tear the nucleus in order to discover whether there were more or fewer number of lobes to the nucleus than was shown by Wright's differential method. Slides were scratched with emery paper and then cleaned and the blood drawn slowly across the scratches so as to catch and tear the neutrophils.

Counts made from such preparations give the same neutrophilic blood-picture as did the smears prepared by the normal Wright's differential method. In

some cases the neutrophil was stretched to several times its normal length, but in all such cases the nucleus stretched also and the number of lobes remained the same.

These experiments demonstrate that the number of lobes of the nucleus is not an artifact due to any one stain, but is a definite state taken by the nucleus when killed, which must indicate some definite differences in the state of the nucleus of the different living neutrophils.

3. Normal Blood-Picture in Man and in Guinea-Pigs: (a) Smears were prepared from over twenty different persons whose condition was, as far as known, normal. These different blood-pictures gave indices which varied slightly from each other and yet varied within a small range, which could be called normal. This blood-picture corresponded very nearly with that of Arneth, which was taken from a large number of subjects.

(b) Smears were obtained from a few subjects at different times of day for a period of time extending over a week or several weeks and in two cases extending over a period of two years. The results in these cases show indices which vary, but not definitely enough to be attributed to any one cause, and the amount of the index variation in each case was so slight as still to come within the limits of the normal blood-picture, except in the case of one of the subjects studied for a period of two years, in which the blood-picture shifted decidedly, owing to appendicitis.

These observations showed that the average normal blood-picture was: I/5, II/26, III/36, IV/28, V/5: leukocytes, 9,000; index, 49:51, but that any blood-picture with an index between 45:55 and 55:45 may be called normal unless one of the classes should be in some way most unusual.

(c) Ten guinea-pigs were studied for a period of over two weeks. Smears were prepared from the blood of these animals every two hours during the day. The result gave a picture comparable to that of the human blood, except that the number of the lobes of the nucleus of the neutrophil is greater in the guinea-pig than in man, and the index is therefore different. The normal guinea-pig neutrophilic blood-picture is as follows: I/5, II/20, III/38, IV/30, V/6, VI/5: leukocytes, 10,000; index, 42:58.

The blood-picture as shown by the guinea-pig varies more for the individual animal than it did for the individual person, so that in all experiments with guinea-pigs, the animals were kept for a week or ten days and the normal blood-picture established for each animal before the experiment was begun.

4. Effect on Neutrophilic Blood-Picture of Subcutaneous Inoculation of Virulent Culture of Tubercle Bacilli: Guinea-pigs were inoculated subcutaneously with varying amounts of an emulsion of living tubercle bacilli in physiological salt solution. The blood-picture in all cases was very much like the following, except that the number of days necessary to reach the typical tuberculosis blood-picture varied with the amount used.

Table 2.—Blood-Picture of Guinea-Pig VI, Inoculated Three Million Strong Emulsion of Tubercle Bacilli.

Time with Reference to Inoculation.	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	Leukocytes.	Index.
Before	3	21	32	72	6	5	..	..	9,000	40:60
At time of	4	19	36	30	6	5	..	..	9,600	41:59
3 hours after	2	14	28	34	12	10	..	..	10,000	30:70
12 hours after	2	4	16	34	20	10	10	5	10,200	13:87
24 hours after	2	5	18	36	20	11	6	2	15,000	16:84
48 hours after	3	9	18	38	16	10	6	..	16,400	21:79
4 days after	3	12	38	34	8	3	2	..	14,000	34:66
8 days after	4	18	38	30	8	2	..	..	13,200	41:59
12 days after	8	22	39	24	5	2	..	..	13,400	49:51
18 days after	16	38	40	6	..	..	..	..	13,200	74:26
21 days after	23	45	32	..	..	..	..	..	12,800	84:16
25 days after	24	53	23	..	..	..	..	..	13,000	87:13

When the animal was killed the lungs and liver were found to be full of tubercles.

These experiments seem to show that while the first result of the inoculation of the tubercle bacilli is to increase the number of neutrophils with the greater number of lobes in the nucleus, the prolonged effect of the bacilli is to use up the neutrophils which have the greater numbr of lobes to the nucleus and leave only those with the fewer number of lobes. The individual whose circulating blood contains an increased number of neutrophils with one or two lobes of the nucleus and a decreased number of neutrophils with four or five lobes is probably not in a state of resistance to the bacteria.

5. **The Effect of the Inoculation of Very Small Amounts of Emulsion of Living Tubercle Bacilli:** A number of guinea-pigs were inoculated with such a small amount of the bacilli that when killed after several months no trace of tuberculosis was found in the organs of the animal. In these cases the blood-picture showed a very slight increase in the number of lobes of the nucleus following the inoculation. Then the blood-picture either rapidly returned to normal and remained normal or remained with a slight increase in the number of lobes of the nucleus.

6. **Blood on Agar with Tubercle Bacilli:** A drop of fresh flowing blood was placed on an agar plate and at one edge of the drop was placed a very small amount of tubercle bacilli. The agar plate was kept in the incubator and smears were made every five minutes from the side of the drop of the blood near the bacilli as well as from the side of the drop away from the bacilli. The blood-picture showed an increase in the number of lobes of the nucleus of the neutrophils of the blood, which touched the bacilli, even after a period of five minutes, while the neutrophils of the blood away from the bacilli remained normal.

Experiments in which washed leukocytes were used instead of normal blood gave the same result.

7. **The Effect of Contamination:** (a) Blood was drawn from the end of a finger purposely made dirty and slowly smeared on slides which had been handled and were not clean. In all cases such smears gave a neutrophil blood-picture in which the number of the lobes of the nucleus had increased.

(b) Blood was drawn from a clean finger and placed in small glass chambers which were not sterile and which were left open to the air of the incubator. Smears prepared from such blood after a period of five or ten minutes

showed a decided increase in the number of lobes of the nucleus. Neutrophils with as high as ten or twelve lobes to the nucleus were present, while the blood from the same finger normally prepared or kept in sterile dishes in the incubator never contained neutrophils with a nucleus of more than five lobes.

(c) One of the guinea-pigs inoculated with 2,000,000 weak emulsion of tubercle bacilli developed an open abscess at the point where the needle entered. The smears prepared from the blood of this animal gave the following blood-picture, which shows an irregularity as well as an increase in the number of lobes of the nucleus of the neutrophils.

Table 3.—Blood-Picture of Guinea-Pig II, Which Developed Abscess Following the Inoculation of Two Millions of Tubercle Bacilli.

Time with Reference to Inoculation	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	Leukocytes,	Index.
Before	4	21	36	30	5	44	..	..	10,000	43:57
At time of	3	21	35	30	6	5	..	..	10,200	41:58
3 hours after	2	16	32	30	8	6	3	3	12,000	34:66
12 hours after	2	14	30	20	14	10	6	4	14,000	31:69
24 hours after	3	20	30	18	13	7	5	4	13,500	38:62
48 hours after	2	21	32	26	10	6	3	..	14,000	39:61
4 days after	3	14	30	26	11	10	4	2	18,000	32:68
8 days after	1	10	33	28	13	7	5	3	16,000	27:72
12 days after	..	4	24	30	15	10	10	7	15,000	16:84
16 days after	12	24	30	20	10	4	..	..	22,000	51:49
18 days after	..	6	16	32	23	13	5	5	16,000	14:86
20 days after	4	10	18	34	19	11	4	..	17,000	23:77

Animal was killed—liver and lungs contained many tubercles.

8. The Neutrophil Blood-Picture in Opsonic Index Work: Many of the slides studied were obtained through the courtesy of Dr. W. H. Park of the New York Department of Health and others were prepared by us. An emulsion of dead bacilli was used and the pipets were left in the incubator for fifteen minutes.

All such smears gave a blood-picture in which the number of the lobes of the nucleus of the neutrophil was greatly increased. Neutrophils with six, eight, ten, twelve and in one case sixteen lobes to the nucleus were found, while almost no neutrophils with one, two or three lobes were present.

9. Effect of Inoculation with Snake Venom: Inoculation of guinea-pigs subcutaneously with snake venom gave a neutrophilic blood-picture in which the number of lobes of the nucleus was decidedly increased. Many neutrophils contained nuclei of so many lobes that they had the appearance of rosettes. The neutrophils of the bone-marrow showed the same change in quite as great an extent as did those of the circulating blood.

Conclusions From Experiments.

1. The neutrophil is an organism which reacts quickly and definitely to its environment and this reaction is indicated by the number of lobes of the nucleus.

2. The presence of neutrophils which have a nucleus of a greater number of lobes than normal indicates some reaction to a change in the environment such as the presence of a toxin or of bacilli.

3. The presence of neutrophils which have a nucleus of a less number of lobes than normal indicates either that the neutrophils are being used up and so only those in the younger stage are left in the circulating blood or that the

neutrophils entering the blood fail to undergo the metabolic or morphologic change which causes the increase in the number of lobes of the nucleus and so remain in the one or two lobe form.

4. The index shown by the neutrophilic blood-picture shows the condition of the neutrophils of the blood and in this way gives an insight into the condition of the subject.

Pulmonary Tuberculosis.

Our observations in pulmonary tuberculosis were made on seventy-eight cases with a total of 315 separate blood-counts. The period of time covered by our studies was three years, and this has made it possible to follow many cases of tuberculosis over a long period, which is very important in any effort to draw conclusions from observations made in regard to this disease.

We wish to emphasize strongly the importance of studying the changes of the blood according to the clinical course of the disease and not according to the pathological conditions in the lungs, as has been done by the majority of observers who have reported on the changes in the leukocytes in tuberculosis, many of whom have followed the classification of Grawitz.

We have classified our cases according to the stages of the National Association, and in addition to that, according to the clinical prognosis at the time of the first observation, and have followed these cases into their subsequent history, endeavoring to coordinate our blood findings with the clinical course.

The Number of Leukocytes.

The majority of authorities state that the leukocytes are not changed in uncomplicated tuberculosis, but are increased when secondary infection occurs, resulting in caseating lesions or cavity formation, or with exudative inflammation or in some of the complications, such as hemorrhage and enteritis.

Kjer-Petersen (5) made a particularly careful study of the number of leukocytes in tuberculosis. He comes to the conclusion that the extent of the lesion has no relation to the number of leukocytes; that in men in early stages the number is normal if there is no fever, and in third stage cases with no fever moderately increased but very variable—6,000 to 15,000.

In febrile cases there is some increase in all stages, and particularly those of continuous fever, which he attributes to mixed infection. Other cases of pure tuberculosis with continuous fever are very variable but average low counts. In women, as already stated, he found the count very variable.

Stein and Erbmann (38), Kjer-Petersen (5), Applebaum (39) and Steffen (40) think a sudden increase in leukocytes important evidence of cavity formation. Richard (41), Benzançon, de Jong and de Serbonnes (42, 43) and Halbron (44) and Steffen (40) emphasize the importance of following the clinical course regardless of the pathology, and found a tendency to leukocytosis with the exacerbations of the disease.

Claude and Zaky (45) obtained similar results in guinea-pigs, the leukocytes in the controls being increased and in treated animals less so. Ullom and Craig (46) state that a decrease in the number of leukocytes in advanced cases is unfavorable. Arneth also looks on a leukocytosis as favorable and states that a shifting to the left of his blood-picture is worse if the number of the leukocytes is low. Craig (47) disagrees with Stein and Erbmann (38) in the significance of the increase of leukocytes with cavity formation. They found often no leukocytes in cases of cavity.

Our own results in the enumeration of the total number of leukocytes coincide closely with those obtained by other observers, except that the average

obtained in incipient cases and in the non-tuberculous cases is a little higher, 9,874 and 10,209 respectively. There is a distinct increase in the number of leukocytes with the progression of the disease. This is graphically shown in Chart 1. The striking difference between the non-tuberculous (10,209) and the moribund patients (26,300) corresponds with the reports of other observers of the frequent marked leukocytosis in dying patients. The variations according to the stage, the clinical prognosis at the time of observation and the subsequent course of the disease show a distinct relationship between these factors and the number of leukocytes, which the chart shows better than description. The fact that the figures shown for the prognosis correspond quite closely with those for the subsequent history, would appear to make the enumeration of the leukocytes in tuberculosis a procedure of more prognostic value than it is usually considered.

Inasmuch as our cases have been followed in the majority of instances for more than two years since the blood-counts were made, the data presented as to the subsequent history represent accurately the course of the disease.

Table 4.—Blood-Counts in Various Classes of Tuberculous and Non-Tuberculous Cases.

	No. of Cases.	No. of Observ.	Aver. No. Leuk.	Per Cent. Neut.	Aver. Arneth Indices.	Per Cent. L. L.	Per Cent. S. L.	Per Cent. Eos.	Per Cent. Bas.
Not tuberc.	9	37	10,209	64.4	54:46	17.5	11.8	5.8	0.53
Pulmon. tuberc:									
Incipient	15	50–54‡	9.874	72.4	64:36	14.4	9.0	3.7	0.52
Advanced	30	91–114	10,200	74.0	67:33	15.2	7.9	2.4	0.52
Far adv.	20	91–101	13,218	77.9	75:25	13.9	6.2	1.5	0.44
Dying from tuberc. other than pulmon.*	4	8–9	26,300	78.5	66:34	17.1	4.1	0.29	0
Prognosis:									
Good	19	62–79	9,398	71.8	60:40	14.8	9.1	3.7	0.53
Doubtful	18	77–87	11,591	78.0	73:27	13.7	5.8	2.0	0.32
Poor	29	84–99	12,486	77.1	72:28	13.4	7.5	1.6	0.35
Subsequent hist. of tuberculous patient:									
Improved	17	60–74	9,323	71.6	60:40	14.9	9.9	3.0	0.56
Dis. station.† ..	3	18	12,486	79.1	72:28	13.3	5.6	1.6	0.43
Dis. progres. ..	26	102–121	13,897	77.3	74:26	14.5	5.9	1.86	0.43

*Tuberculosis of kidney, of bladder, of psoas muscle, and acute miliary.

†Two of these cases were far advanced chronic cases.

‡Complete counts for every point were not invariably made.

Polynuclear Leukocytes.

Almost all observers have found that when cases of tuberculosis showed a leukocytosis, it was usually of a polynuclear form and it is explained by the incidence of secondary infection. Experimentally, Achard and Loper (48) found that there was a primary increase in the polynuclears followed by an increase in the mononuclears.

Claude and Zaky (45) also found that the percentage of polynuclears was much less in treated animals with a tendency to localized lesions than in the control animals. Benzançon, de Jong and de Serbonnes (42) consider a polynucleosis as significant of acute and active disease and of great value in both prognosis and treatment, stating that all active treatment, such as tuberculin,

arsenic, superalimentation, etc., is contra-indicated if polynucleosis is present. They consider it a guide to the dosage of tuberculin similar to the opsonic index. Almost all observers consider an increased percentage of polynuclear an unfavorable sign.

In our own studies the percentage of neutrophils varies from 64.4 per cent. in the non-tuberculous to 78.5 percent. in the moribund patients, and in general the percentage is higher the more advanced the disease and the more unfavorable the prognosis and subsequent course. All of these percentages are high in our observations, even the incipient cases showing a percentage of 72.4 per cent.

The increase in the more unfavorable cases, however, is very marked and would seem to corroborate the observations of others that the leukocytosis of tuberculosis is due to an increase in the polymorphonuclears. A high percentage of neutrophils would therefore seem from our observations to indicate an unfavorable prognosis, and this would be the more probable were an actual leukocytosis also present.

Arneth's Blood-Picture.

As already stated, unfavorable clinical reports on the value of Arneth's blood-picture in tuberculosis have been made by Paulicek (20), Bourmoff and Brugsch (21), Solis Cohen and Strickler (23) and Kagan (22). Many other observers, particularly those who have reported more recently, corroborate Arneth's findings in this disease. Among them may be mentioned Klebs (31), Sabrazès (36), Dluski and Rospedishowski (32), Pottenger (49), Von Bonsdorf (35), Bushnell and Treuholtz (37), and Minor and Ringer (33).

We have already described our method of making the Arneth count and the meaning of the indices recorded in the tables. While we appreciate the fact that the use of these indices is a marked departure from Arneth's own method, we have found from experience that they represent very accurately the main contention, i. e., the shifting to the left in the nuclear blood-pictures. It has been impracticable to tabulate the details of these counts and for that reason only the indices appear and the actual percentage of cells with their different number of nuclear segments is not represented.

We feel that this in no way detracts from the practical value of the results presented, and we are anxious to emphasize our appreciation of these indices, or similar ones, as a valuable addition to the information that may be obtained from the differential leukocyte count. The more cumbersome tables of Arneth would never come into general use, but their chief clinical value is retained in the indices which, because of their definite numerical form, can easily be incorporated into the results of every leukocyte count and probably will be, as soon as their value is more generally appreciated.

Our results are seen in Chart 1, the left-hand number of the index being employed in the chart.

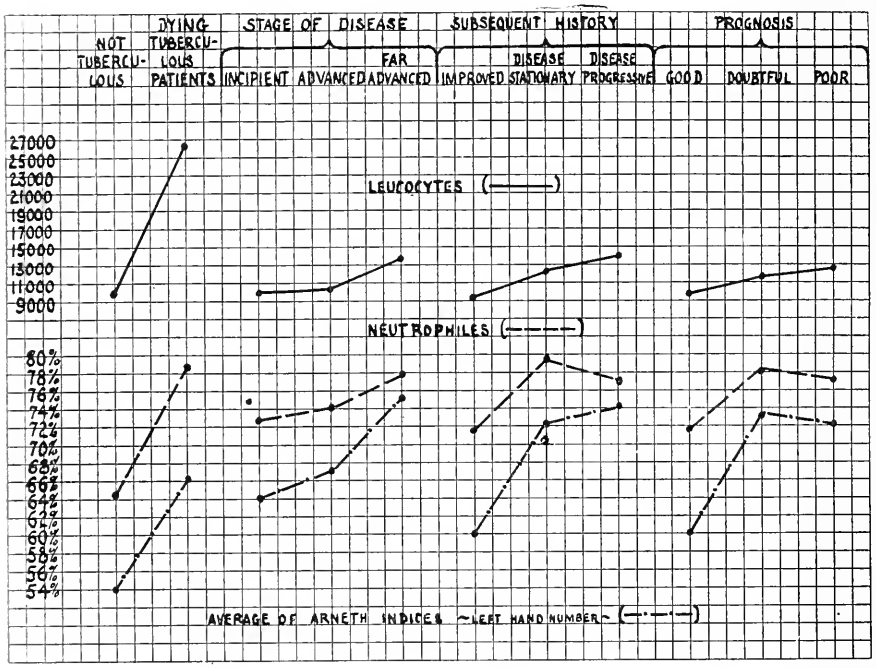
Our average index for the non-tuberculous cases was 54 : 46, and this corresponds closely with those obtained in a considerable number of observations on normal individuals. In all other cases it will be seen that the left-hand number is much larger, and this represents the number of cells with one and two nucleus segments, plus one-half of the number having three segments. It is evident how clearly the shifting to the left is demonstrated in our observations.

The degree of shifting also becomes more marked as the cases are more advanced until an average index of 75 : 25 is noted in the far advanced cases. Corresponding to this is the marked shifting noted in the cases with unfavorable prognosis and the subsequent clinical course.

The relatively slight shifting in the four cases of moribund patients is sur-

prising. It is not to be expected from the progressive shift observed in cases with unfavorable course during the period of observation. While it may be that there is a change in the nuclear blood-picture shortly before death, the number of cases of this sort which we have studied is so small that no definite deductions should be drawn from them.

CHART I



From the general standpoint of diagnosis and prognosis, however, we feel that in the study of the nuclear blood-picture, we have obtained information which is of the greatest value in tuberculosis. The changes noted are more constant than are any of the others in the blood and much more so than are many of the usual clinical signs and symptoms.

In diagnosis the field of usefulness is limited because of the comparatively slight shifting in early cases, and the fact that it may occur in other conditions in which the general condition of the patient is below normal, our experience and that of others being that the shifting to the left is simply a response to any deleterious influence in the body, especially the bacterial infections.

In prognosis, however, both at the time of original examination and during the course of the disease, the degree of shifting to the left is an exceedingly sensitive indication of the resisting powers of the individual and of the progress of the disease.

We have found that patients with very slight lesions but persistently presenting an unfavorable nuclear picture almost invariably do badly, and that, on the contrary, those with extensive lesions but fairly good nuclear picture show a marked tendency to resist the disease successfully. Moreover, during the clinical observations of a case, its real progress in one direction or the other can

usually be more accurately anticipated by the changes in the nuclear picture than by any other clinical means.

Even clinical apparent cures, as Arneth well claims, are insecure unless the nuclear picture has come nearly to approach the normal, and our experience has made us very reluctant to advise a return to work and less favorable surroundings in such cases, while such return may be allowed with considerable confidence when a healthy blood-picture is found.

Arneth's Blood-Picture as a Guide to Specific Treatment.

Arneth asserts that his blood-picture is of great value both as a guide to dosage in tuberculin and also as a test for the effect of the treatment. He also considers it of value in deciding the question of the necessity for secondary courses of treatment with tuberculin. Uhl (50) has found it of value in a similar way. Von Bonsdorf also used it as a guide for dosage, and notes a negative phase in the blood-picture after tuberculin similar to that of the opsonic index. Arloing and Genty (51) have compared the blood taken with the agglutinating power of the serum and also with the results of treatment with Maragliano's and Marmorek's serums and find a general correspondence in the results. Rover (52) also corroborates these results with Marmorek's serum. Kaufmann (53), on the contrary, found no relation between the effects of Marmorek's serum or the clinical course and the changes in Arneth's blood-picture.

In our own work we at first attempted to use Arneth's blood-picture as a guide to tuberculin dosage, and for a while it seemed as though results were encouraging; but later experience has demonstrated to us that it is of little or no value, and that the usual clinical guides are much more consistent and delicate than any changes we have found in the leukocytes.

The Lymphocytes.

In a general way, the majority of observers have noted a change in the lymphocytes inversely proportionate to the neutrophils. In consequence, a diminishing number of lymphocytes would be considered unfavorable and an increase correspondingly favorable. Da Costa (10) asserts that occasionally the leukocytosis of advanced disease is due to lymphocytes. Warthin (54) also noted this symptom in chronic cases, as does Cabot (6). Arloing and Genty (51), Richard (41), Benzançon, de Jong and de Serbonnes (42), Halbron (44), Sabrazès (36), Steffen (40), Ullom and Craig (46) all consider that an increased percentage is favorable. Steffen (40) and Bezançon, de Jong and de Serbonnes (42) lay especial stress on its favorable significance during periods of improvement.

Experimentally, Achard and Loeper (48) found that secondarily in guinea-pigs there was a lymphocytosis following a polynucleosis. Claude and Zaky (45) found that in controls the lymphocytes were diminished and that in chronic cases and treated patients the large mononuclear cells were increased.

Webb and Williams (9) lay stress on the increase of the large mononuclears in improving cases and as an effect of altitude. They think that the beneficial effects of altitude in tuberculosis may be attributable to this increase.

In our own results we have separated the lymphocytes into the large lymphocytes and the small lymphocytes with the following results:

The Large Lymphocytes.

The proportion of these cells which we have found in our counts is fairly constant in all stages and all degrees of progression of the disease. No apparent relationship to the clinical data can be made out from the slight variations in percentages recorded in Table 1 and charted in Chart 2. It would seem,

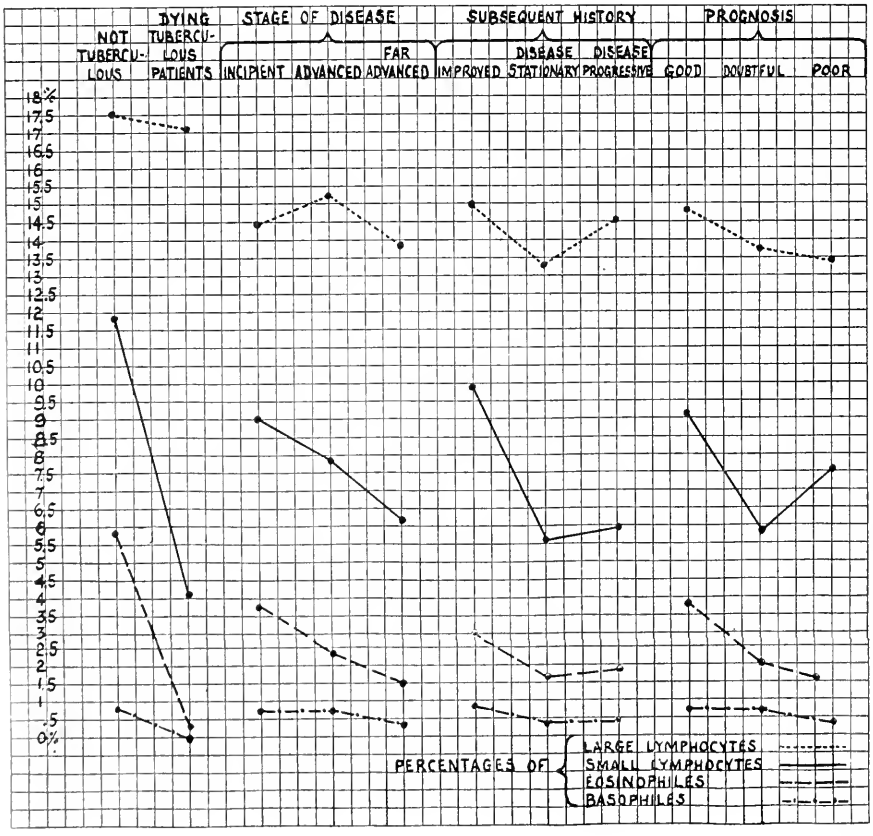
therefore, that no medical importance is to be attached to these cells in tuberculosis.

The Small Lymphocytes.

In sharp contrast to the large lymphocytes a very definite and interesting relationship appears to exist between the small lymphocytes and the clinical manifestations. This is apparent in Table 4 and is shown very graphically in Chart 2. With the greater extent of the lesion and the unfavorable progress of of our cases there is a progressive diminution in the percentage of small lymphocytes. In the incipient cases, those with good prognosis, those whose subsequent history show improvement and in the non-tuberculous cases the percentage of the cells is found to be uniformly high, varying from 9.0 per cent. to 11.8 per cent. On the other hand, the more advanced and unfavorable cases show percentages from 4.1 per cent. to 6.2 per cent.

The moderately advanced cases and those with doubtful prognosis standing midway between the other classes 7.5 per cent. to 7.9 per cent. It is evident that in our cases the increased percentage of the neutrophils in unfavorable cases has been largely at the expense of the percentages of the small lymphocytes.

CHART II



The Eosinophils.

A good deal of emphasis has been laid on the role of the eosinophils in the circulating blood as a guide to the clinical condition of the cases of tuberculosis. In general, in acute progressive cases with secondary infection, the eosinophils have been observed to be diminished or absent and to reappear or increase during the periods of improvement or in early and favorable cases. Such is the opinion expressed by Naegeli (4), Ewing (7), Swan (55), Holmes (56), Arloing and Genty (51), Sabrazès (36), Cabot (6), Da Costa (10), Zappert (57), Galbraith (58), Applebaum, Richard (41), Steffen (40) and others. Bezançon, de Jong and de Serbonnes (42) think that the presence of the eosinophils indicates good resisting power and note their increase in improving cases especially in chronic inactive cases in which they consider that the tubercle bacillus lives in the body as a parasite.

Holbron (44) asserts that they are diminished in advanced cases and absent in all cavity cases. Neusser (59) also considers that the increase in eosinophils denotes an immunity to the tubercle bacillus.

Webb and Williams (9) noted a very slight decrease in advancing cases which, however, was not distinct. Ullom and Craig (46), Solis Cohen and Strickler (22) and Craig (47) found no relation between these cells and the clinical course of the disease. Experimentally, Claude and Zaky (45) and Achard and Loeper (48) found that these cells were diminshed in control cases, but reappeared subsequently if the animals lived, and in chronic cases they were increased.

Numerous authorities, including Ewing (7), Cabot (6), Bischoff (60), Rieder (61) and Bodkin (62), note an increase in the eosinophils after the administration of tuberculin.

Our results coincide with the majority of observers, and we have found that with the progression of the disease the cells become less and less numerous until they almost entirely disappear from the blood in the terminal stages.

Our results show 5.8 per cent. in non-tuberculous cases and 3.7 per cent. in the incipient cases and those with good prognosis. This percentage is reduced to 1.5 per cent. in the far-advanced patients and 0.29 per cent. in the moribund patients. Chart 2 shows this relationship very clearly.

The Basophils.

We have found no reference in literature as to the behavior of the basophil leukocytes in tuberculosis. The small number of these cells found in the blood renders valueless conclusions drawn from the count of from only 100 to 200 cells. The indications, however, are that they do not vary appreciably in the various phases of tuberculosis and that they have no clinical significance.

Composite Tuberculosis Blood-Picture.

In order easily to appreciate the clinical significance of our findings, we have arranged them in the form of composite blood-pictures for each of the more important features of the disease, in Table 5.

Table 5.—Blood Counts in Various Classes of Tuberculous and Non-Tuberculous Cases.

I. Non-Tuberculous Cases.

Nine cases—thirty-seven counts:

Total Number	N.	L. L.	S. L.	E.	B.	Index
10,209	64.4	17.5	11.8	5.8	.53	54 : 56

II. Incipient Tuberculosis.

Fifteen cases—Fifty-four counts:

Total Number	N.	L. L.	S. L.	E.	B.	Index
9,874	72.4	14.4	9.	3.7	.53	64 : 36

III. Advanced Tuberculosis.

Thirty cases—114 counts:

Total Number	N.	L. L.	S. L.	E.	B.	Index
10,200	74.	15.2	7.9	2.4	.52	67 : 33

IV. Far-Advanced Tuberculosis.

Twenty cases—101 counts:

Total Number	N.	L. L.	S. L.	E.	B.	Index
13,218	77.9	13.9	6.2	1.5	.44	75 : 25

V. Moribund Patients.

Four cases—nine counts:

Total Number	N.	L. L.	S. L.	E.	B.	Index
26,300	78.5	17.1	4.1	.29	.0	66 : 34

VI. Cases with Good Prognosis.

Nineteen cases—seventy-nine counts:

Total Number	N.	L. L.	S. L.	E.	B.	Index
9,398	71.8	14.8	9.1	3.7	.53	60 : 40

VII. Cases with Poor Prognosis.

Twenty-nine cases—ninety-nine counts:

Total Number	N.	L. L.	S. L.	E.	B.	Index
12,486	77.1	13.4	7.5	1.6	.3	72 : 28

VIII. Cases Which Showed Subsequent Improvement.

Seventeen cases—seventy-four counts:

Total Number	N.	L. L.	S. L.	E.	B.	Index
9,323	71.6	14.9	9.9	3.0	.56	60 : 40

IX.—Cases Which Subsequently Did Badly.

Twenty-six cases—121 counts:

Total Number	N.	L. L.	S. L.	E.	B.	Index
13,897	77.3	14.5	5.9	1.86	.43	74 : 26

Pneumonia.

A review of the literature concerning the behavior of the leukocytes during lobar pneumonia shows that the presence of leukocytosis in this disease has been recognized from the very beginning of blood examinations. Cabot (6) states that the leukocytosis when marked indicates good resistance on the part of the individual or a severe infection, and that a slight leukocytosis or normal leukocyte count indicates a poor resistance or a mild infection. He considers the absence of leukocytosis a very bad sign but its presence of no significance for either good or bad. In 842 cases ninety showed no leukocytosis and of these eighty-three were fatal.

Cabot's ideas are similar to those expressed by Ewing (69), Naegeli (4), Da Costa (10), Buchanan (63), and others. Türk (2) states that the leukocytosis bears no relation to the severity of the disease, and that if it continues after crisis it indicates delayed resolution or some complication.

Rieder (61) states that it has no relation to the height of the fever. Von Limbeck (64) states that the leukocytosis is in proportion to the lung involvement. This contention has been disproved by numerous observers. Von Jaksch (65) made a careful study, showing the importance of leukocytosis from

the standpoint of prognosis and advocated artificial means to produce leukocytosis such as drugs, local abscess formation, etc. These ideas are now obsolete.

Experimental Evidence.

Tschistovitsch (quoted by Von Limbeck) (64) experimented with guinea-pigs, and found that an injection of highly virulent cultures of pneumococcus produced no leukocytosis and the animals died. Weaker cultures produced leukocytosis and the animals recovered.

Rieder (61) inoculated guinea-pigs intraperitoneally with pneumonia sputum and obtained subnormal leukocyte count and subnormal temperature. Williamson (66), by injections of pneumococcus cultures in guinea-pigs, obtained a leukocytosis of varying intensity followed by a diminution in the number of leukocytes. He also noted no relation between leukocytosis and the course of the disease.

Clinical Study.

Our studies of the leukocytes in pneumonia have failed to reveal the definite clinical relationships that we found in tuberculosis.

Forty cases were studied and on these 131 separate blood counts were made. Almost the entire number were cases of lobar pneumonia, but a few of bronchopneumonia are included. All of the observations were in adults.

In attempting to trace the connections between the blood counts and the clinical findings, we have analyzed the cases according to the extent of involvement, the clinical severity of the infection, the method of defervescence, the duration, the final results and the complications.

Our results in their relation to all of these factors are tabulated in Table 6.

Table 6.—Observations in Lobar and Bronchopneumonia During the Acute Attack.

	No. of Cases	No. Observ.	Aver. No. Leuk.	Per Cent. Neut.	Aver. Arneth Indices.	Per Cent. L. L.	Per Cent. S. L.	Per Cent. Eos.	Per Cent. Bas.	Per Cent. Myelo
Involvement:										
One lobe ..	24	91-92	17,058	77	67 : 33	16.8	4.6	1.2	0.27	0.11
Two lobes.	16	35-39	18,012	76.5	56 : 44	17.6	4.1	1.5	0.23	0.04
Severity:										
Mild	11	47-49	17,634	76.9	66 : 34	16.5	4.6	1.6	0.25	0.09
Moderate .	13	45-46	14,532	72.9	63 : 37	19.5	5.3	1.8	0.42	0.08
Severe	11	28-29	20,250	81.8	62 : 38	15	3.0	0.08	0.3	0.07
Defervescence:										
Crisis	21	71-74	16,355	74.9	64 : 36	17.6	5.2	1.9	0.23	0.14
Lysis	8	30-32	17,028	77.5	64 : 36	17.5	3.6	.77	0.48	0
Duration:										
Up to six days inc.	4	10	16,660	72.1	66 : 34	18.7	7.8	1.4	0	0.13
Seven days and over ..	18	17-18	16,912	76.4	65 : 35	17.4	4.5	1.2	0.30	0
Result:										
Cured	12	40	16,265	77.6	63 : 37	16.5	4.9	0.89	0.077	
Died	10	27	21,040	80.9	60 : 40	15.6	3.2	0.12	0.057	0.18
Delayed resolution	11	46-50	15,255	73.3	66 : 34	18.7	4.9	2.4	0.53	0
Complications:										
Empyema .	3	10	16,600	85.6	61 : 39	11.3	3.0	0.008	0	0
Pericarditis, etc.	6	10-11	17,960	72.8	66 : 34	22.5	3.7	0.66	0.26	0

After Subsidence of Attack.

Resolution:

Good	11	20	12,930	73.0	59 : 41	19.2	6.2	1.4	0.10	0
Delayed ...	6	20-22	12,995	64.4	64 : 36	23.3	7.3	3.93	0.77	0.25

In addition, the observations on the leukocytes after the acute infection was over have been studied in respect to the promptness of resolution in the lungs and these results are also included in the table.

Total Number of Leukocytes.

The usual leukocytosis expected in pneumonia was present in our cases but was not as marked as is often seen. The number of leukocytes varied from 15,000 to 20,000 in all classes of cases excepting in the series of ten patients who died, in which the average of twenty-seven counts was 21,040. This is noteworthy because these results disagree with the more usual finding that a high leukocytosis indicates a good prognosis and a low one the reverse. Our series is too small to enable us to draw definite conclusions in this regard but the results are nevertheless suggestive.

The usual diminution in leukocytosis is noted after the defervescence. This is not so very marked, the counts averaging about 13,000. Most of these counts were made in the first week after defervescence and indicate a gradual return toward the normal leukocyte count.

In order to ascertain whether there was much variation in the leukocytosis of pneumonia in various years corresponding to the rather general opinion that there is a marked difference in the virulence in this disease at different times, we have collected the records of the leukocyte count in lobar pneumonia for five years, 1904 to 1908 inclusive, of the First Medical Division of Bellevue Hospital. Thus 514 cases are studied and the results are tabulated in Table 7.

Table 7.—Leukocyte Counts in Lobar Pneumonia During Acute Illness.

Patients Who Recovered.

Counts Between	1904	1905	1906	1907	1908
	%	%	%	%	%
0-10,000	5	15	7	7	13
11,000-20,000	41	45	33	58	52
21,000-30,000	30	38	43	28	20
31,000-40,000	22	2	15	7	13
41,000-50,000	2	0	2	0	2
Av rage Number of Leukocytes	22,501	18,375	22,865	19,243	19,208

Patients Who Died.

0-10,000	17	10	25	16	11
11,000-20,000	46	53	47	51	54
21,000-30,000	25	25	18	25	28
31,000-40,000	4	12	8	8	7
41,000-50,000	8	0	2	0	0
Average Number of Leukocytes	19,652	19,938	18,216	18,024	18,218

Summary for the Five Years of 514 Cases.

	Recovered.	Died.	Recovered.	Died.
	Number		Per Cent.	
0-10,000	26	39	10	16
11,000-20,000	123	123	46	50
21,000-30,000	85	59	31	24
31,000-40,000	32	19	12	8
41,000-50,000	3	5	1	2
Total	269	245	100	100
Average Number of Leukocytes	20,444	18,827		

From this table it does not appear that there is any great variation in the various years, certainly none from which any valuable inference could be drawn.

These results also do not demonstrate any definite relationship between fatality and low leukocyte counts. The average for the entire 514 cases showed 20,444 for the patients who recovered and 18,827 for those who died. These cases are all uncomplicated and the counts of course were made by numerous observers.

Differential Count.

Cabot (6), Ewing (7), Da Costa (10), Türk (3), Rieder (61) and others all find that the leukocytosis of pneumonia is due to the increase of the neutrophils. Türk, who has studied the differential count in pneumonia most thoroughly, states that the neutrophils are increased in proportion to the leukocytosis; that they fall with the crisis but not with a pseudocrisis; that the large mononuclear cells and transitionals vary with the neutrophils; that the lymphocytes may be increased, normal, or diminished in number but that after a crisis they are usually increased; that eosinophils are absent or few during the fever but that their presence is favorable and that after a crisis they are distinctly increased, their reappearance in the blood often being evidence of beginning resolution. Myelocytes may appear if the infection is very acute.

Most authorities agree in general with Türk's observations. Becker (67) states that he has never found any eosinophils present in fatal cases. Loeper (68) states that if the percentage of neutrophils is over 90 per cent. the case is almost invariably fatal.

Our observations show that the neutrophils are invariably increased excepting after defervescence; that they vary from 72 to 77 per cent. in all classes of cases excepting in the very severe infections in which they were 81.8 per cent. In patients who died they were 80.9 per cent. and in those who had empyema as a complication, 85.6 per cent.

It would appear, therefore, that an excessive increase in the percentage of neutrophils indicates a very severe infection. In the lymphocytes our studies fail to reveal any clinical evidence of value. The percentage of large lymphocytes varies from 15 to 23.3 per cent. and the small lymphocytes from 3 to 7.3 per cent.

In the cases of severe infection the percentage of large lymphocytes was comparatively low and higher in those with delayed resolution after defervescence.

In the eosinophil cells the blood counts during the acute illness showed nothing of note, the percentage being normal throughout. In six cases of delayed resolution, however, the average percentage of these cells in twenty counts was 3.93. This moderate increase is in accord with the results of other

observers in such cases and appears to have some definite relationship to the condition in the lung.

The basophils and myelocytes were observed in very small numbers and are apparently of no clinical significance.

The Arneth Blood-Picture.

Very few studies of Arneth's blood-picture of pneumonia have been reported. Arneth reports on sixteen cases and notes greater variations in his blood-picture between different cases of pneumonia than in any other infectious disease. There is regularly a marked shifting to the left which may come immediately with the onset or may come on gradually and increase to its highest point just before or after crisis.

Complications, according to Arneth, bring out a new and increased shifting to the left. The blood-picture may return to the normal quickly, corresponding to a rapid resolution, or may continue to get worse at the time of the crisis, even though the total leukocyte count may fall.

Cases defervescing by crisis are more apt to return to normal quickly and those by lysis more slowly. In delayed resolution and chronic interstitial pneumonia, a shifting to the left with a normal or diminished leukocyte count may persist for months. Arneth draws no clinical conclusions of value from his own results.

The observations on our cases show a moderate shifting to the left in the Arneth blood-picture to be a constant factor in our cases of pneumonia. This shifting is less marked than in the more advanced cases of tuberculosis and does not vary in any definite relation to the clinical aspects of the cases studied, the index varying from 60 : 40 to 67 : 33.

Very quickly after defervescence the index tends to go back to the normal if good resolution is established. The average index of all such cases studied was 59 :41; but these counts were made usually within the two or three days after defervescence, and we have noted in some cases a return to absolute normal nearly coincident with the signs of complete resolution in the lungs.

In the six cases of delayed resolution, however, the average index was 64 : 33, showing the continuation of the shifting to the left in such cases after the fever had subsided, the whole picture in such cases being very similar to that of moderately advanced tuberculosis.

It is to be noted that the least shifting to the left of any clinical group of cases was in the ten patients who died, the average index being 60 : 40.

An unusual average index is noted in Table 2, under the group of sixteen cases with involvement of two lobes.

This average does not accurately represent the usual condition of affairs in such cases, but in this series is markedly modified by several observations on one case, which showed a very unusual blood-picture with a marked shifting to the right. This patient died and autopsy showed a marked purulent bronchitis in addition to the unresolved pneumonia.

The index above noted, 60 : 40, for the patient who died, is also influenced by this case, which cannot be considered at all representative of the usual blood-picture found in pneumonia.

This case is deserving of further consideration as tending to corroborate our experimental observations, that in the presence of a purulent infection the shift may be to the right and the fragmentation of the nucleus becomes very marked in a considerable number of the neutrophils, even up to seven or eight segments.

In general, we have found the determination of Arneth's blood-picture of practically no clinical value in pneumonia.

The composite blood-picture of pneumonia from our results would be:

Total Number.	N.	L. L.	S. L.	E.	B.	M.	Index.
17,058	77	16.8	4.6	1.2	0.27	0.11	67 : 33

The only other composite blood-picture worthy of separate note in our findings are:

After Defervescence with Good Resolution.

Total Number.	N.	L. L.	S. L.	E.	B.	M.	Index.
12,930	73	19.2	6.2	1.4	0.1	00	59 : 41

After Defervescence with Delayed Resolution.

Total Number.	N.	L. L.	S. L.	E.	B.	M.	Index.
12,995	64.4	23.3	7.3	3.93	0.77	0.25	64 : 36

CONCLUSIONS.

Pulmonary Tuberculosis.

1. The study of the leukocytes gives valuable information in the prognosis and clinical course of pulmonary tuberculosis.

2. In diagnosis of incipient cases it is of no assistance but in differential diagnosis of whether more acute pulmonary lesions are due to tuberculosis or some other infection it is sometimes helpful.

3. Arneth's differential neutrophil count is important in tuberculosis.

4. In general, the following changes in the leukocytes occur in cases of pulmonary tuberculosis which are progressively doing badly or are in an exacerbation of the disease:

(a) A leukocytosis.

(b) An increased percentage of neutrophils.

(c) A diminished percentage of small lymphocytes.

(d) A diminished percentage of eosinophils.

(e) A marked shifting to the left of Arneth's blood-picture. Conversely, changes in the opposite direction in any of the above factors are favorable.

Pneumonia.

1. Leukocytosis occurs as frequently in fatal cases as in those of recovery.

2. This leukocytosis is due to the increased number of neutrophils.

3. When this increase of neutrophils is excessive a very severe infection is indicated.

4. Arneth's differential neutrophil count shows a constant shifting to the left in pneumonia, but it bears no relationship to the clinical course of the disease.

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THE USE OF SALICYLATES IN RHEUMATISM.*

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We frequently hear dissatisfaction expressed concerning the treatment of rheumatism with salicylates This dissatisfaction seems to arise from several causes, either because every ache or pain or morbid process which occurs in the muscles or joints will not react promptly to salicylates, or because the patient cannot tolerate the enormous doses administered, or that even in spite of large doses up to tolerance, certain lesions such as the cardiac lesions of true rheumatism, are not controlled or prevented with facility equal to that by which the pain and swelling of the joints are controlled.

Let us consider for a moment the various treatments that were used before salicylates, in 1875 and 1876, were first used for rheumatism. After having tried the enormous bleedings for depletion, and terrific purgation by mercury, and the various antiphlogistic ideas for which veratrum viride, aconite and antimony were given in large doses, and having frankly realized that these did harm rather than good, the profession settled down to either doing nothing at all and treating symptoms as they arose, or giving the so-called alkaline treatment with potassium bicarbonate, which certainly did not harm the patient as many of the other forms of treatment had done, but which, on the contrary, seemed to show a distinctly beneficial effect, for in fact under this treatment there was and still is less cardiac involvement than under any other single uncombined treatment.

To-day, outside of some form of salicylate treatment the alkaline treatment remains the most effective. The men who first tried the salicylate treatment had seen many methods tried and pronounced a failure, and after thorough trial they were forced to the conclusion that the salicylate treatment shortened the period of pain and fever in rheumatism. Maclagen said that the duration of the pain and suffering was the same number of days as formerly it had been weeks.

One great difficulty from which the profession is slowly emerging is the lack of differentiation made between true rheumatism, which will respond to some form of salicylates, and the various other forms of arthritis which occur, as gout, tuberculosis, the infected arthritis accompanying gonorrhea and scarlet fever, rheumatoid arthritis, and the infected arthritis of unknown origin, such as Still's disease, affecting the smaller joints; these are all classed together as rheumatism and often treated alike. "He who diagnosticates well, will cure well," was never more strikingly illustrated than in the benefits of a correct diagnosis in the various forms of arthritis, and the proper treatment for each varying morbid process. Salicylates will not disinfect the pus of a scarlatinal joint. They will not immobilize and clean out a tuberculosis joint. They will not act as a tonic or as an alterative and build up a depleted body and help it overcome the disintegrating processes of arthritis deformans. They may quiet the pain, but they will not subdue the inflammation in the spindle-shaped joints of the fingers in the acute arthritis of unknown origin. They may stop the pain in gout and, with rest in bed and free purgation, the gouty attack pass by, but we cannot expect their full, rapid and beneficial action except in the case of genuine rheumatism.

By rheumatism is meant here that inflammatory process of the larger joints

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which in childhood is so frequently accompanied with cardiac involvement, and in adult life shows some form of cardiac involvement in over 50 per cent. of those affected. Monarticular rheumatism is exceedingly rare, and in true rheumatism the small joints of the fingers and toes are involved with equal rarity. In this article the symptom-complex of the inflammation in the large joints with or without cardiac involvement is alone referred to as rheumatism.

If we confine ourselves to this conception of rheumatism, I am sure that we shall find some form of salicylates which will act beneficially; but simply because salicylates will act beneficially we are no more justified in treating patients suffering from rheumatism in an absolutely routine way than we are in any other disease. The human intestine is not a test-tube, although often treated like one, and a human organism has its distinct tolerances and intolerances for the different forms of salicylates as distinctly as it has for other remedies.

Most patients are easily irritated by salicylic acid and their stomachs soon reject it, yet these same patients will readily take sodium salicylate with no disturbance. Some patients cannot take sodium salicylate, and yet they tolerate oil of wintergreen in large doses and rapidly respond to it. In other patients I have seen salol and salicin act well, when other forms of salicylates would not, and certainly in children and in the aged, salicin is recognized as the form which is the least depressing and from which the most benefit seems to be obtained.

Aspirin has lately come into vogue and is highly praised by some authors, and yet in my own hands the results from it in rheumatism has been very disappointing. Synthetic methyl salicylate is to be avoided for internal use, and should only be used externally, and yet oleum betulae (the oil of sweet birch) contains the methyl salicylate in the natural form, and is one of the most active and broadly tolerated with the least toxic symptoms of all the salicylates, and it seems a generally accepted fact that the natural salicylates are less irritant than the synthetic.

Synthetic salicylic acid is usually formed from phenol, and the orthosalicylic acid is the one used medicinally. The meta-acid is therapeutically inert, and the para-acid is sometimes present to the detriment of the results obtained, for it is distinctly more toxic than the ortho acid. At times all the phenol is not converted into salicylic acid, and one often wonders when reading of the cases of toxic action of collapse with failing respiration, whether it is not a case of badly prepared synthetic compound in which some phenol or para-salicylic acid is causing the damage to the patient. Some salicylic acid may undergo decomposition, total or partial, with liberation of phenol or compound of phenol, and in fact, some samples of salicylic acid smell of phenol. Sodium salicylate is the form in which the salicylates are usually administered, and it is well borne by most people. If given in solution with excessive alkali it is infinitely less irritant to the gastric membrane than when given in powder.

Salol, which is phenyl salicylate, also has a disadvantage of breaking up into salicylic acid and phenol in the intestine, and hence should not be given if the patient is suffering from kidney lesions. This often acts well, and usually in the amount given is non-toxic, but it cannot be pushed to as high doses nor does it seem to be so rapidly absorbed as sodium salicylate. It is less disturbing to the stomach, and some individuals whose gastric mucous membranes are unusually sensitive to sodium salicylate can tolerate salol. Salophen, which is the acetyl paramido phenol salicylate, had quite a vogue at one time, but seems to have fallen into disuse. Like salol, it has the phenol element in

it, and like salol I have seen it rapidly reduce pain and fever in rheumatism when other forms of salicylate could not be tolerated.

Crofton recommends aspirin as the least irritating to the stomach, intestines and kidneys; he believes that this is the best form of salicylate, and declares that it posseses hardly any toxic symptoms. I have seen a few patients in whom aspirin seemed to be most irritant to the gastric mucous membranes, and produced the general toxic symptoms of the salicylates very rapidly in small doses. In my own experience it has not been as satisfactory for the reduction of fever and an improvement in the general condition of the patient as either sodium salicylate or oleum betulae; in severe cases of rheumatism I have failed to get satisfactory results with it. This probably is due to the presence of the acetyl group, which causes this substance to be slowly broken down and thus probably slowly absorbed. There seems to be no question that to obtain rapidly beneficial results we must have a form of salicylate which is acceptable to most gastric mucous membranes, is quickly absorbed, and hence produces its effect quickly, and then is quickly excreted, that there may be no accumulations in the body.

Some years ago Kinnicutt recommended the oil of wintergreen, which is a natural methyl salicylate. This natural oil of gaultheria is no longer on the market, but the oleum betulae or oil of sweet birch is, and it contains the same natural methyl salicylate. This form of salicylate in many ways fulfils the desired objects of which I have just spoken—the rapid absorption, the rapid excretion, and, to most people, a non-irritating effect on the gastric mucous membranes. In those patients with whom it disagrees, I have found it even when disturbing the stomach to cause less often the headache, the ringing in the ears, and the dizziness. It has been my experience that the majority of patients will respond more quickly and with less disturbance when the oil of birch is given them, than they will to synthetic sodium salicylate or the other forms of salicylates mentioned. Besides the advantages just mentioned, it has the further advantage of being a natural salicylate.

Dr. August Seibert of New York has recently recommended the hypodermic method of giving salicylates. The results reported are excellent, and this method may prove of advantage in those patients whose gastric membranes cannot retain salicylates. The following procedure is recommended: In acute rheumatic infection of joints, heart, pericardium, pleura and central nervous system (chorea), 10 c.c. of a 20 per cent. sterilized solution of fresh sodium salicylate to 100 pounds of body weight are injected fifteen minutes after an appropriate cocain solution (gr. ⅛ to 30 drops of water) has been injected under the same spot. If the injections are made earlier than this, the solution will cause pain. This is repeated every twelve hours. In severe cases, with many localizations of the rheumatic process, the dose may be increased to 15 c.c. of the solution to 100 pounds of body weight.

It is considered essential to the success of this treatment that the above doses be used. Smaller doses will be without effect. The effect may be noticed within three hours after the first injection. Joint stiffness, pain, fever and pulse-rate diminish, and the general feeling of the patient improves. If the injections are continued regularly every twelve hours, the improvement also continues. If they are omitted for twenty-four hours in severe cases, the symptoms will grow worse, but in milder cases the improvement may continue. None of the injected patients presented symptoms of heart depression, ear buzzing, or profuse sweats. In the so-called chronic cases, 10 c.c. to 100 pounds of body weight of the following oily solutions are injected every twenty-four hours: acidi salicylici, 10 gm.; olei sesami, 80. gm.; alcohol, pure, 5 gm.; and gum cam-

phor, 5 gm. The effect of the camphor on the heart is especially beneficial in pericarditis and endocarditis. (1)

Some months ago, a member of a well-known firm in New York suggested to me a form of salicylate which seemed to possess the various advantages of which I have just spoken, and to reduce to a minimum the irritating and toxic symptoms from its use. This salicylate is prepared as follows: oleum betulae, the true oil of sweet birch is taken and saponified either by superheated steam or caustic alkali, the resulting methyl alcohol driven off by heat, and the resulting salicylate decomposed by sulphuric acid and washed until free from sulphates; the salicylic acid is then purified by recrystallization. This salicylic acid is taken and mixed with a sufficient sodium bicarbonate in excess, and with a sugar binder is made into tablets, so that on solution each tablet will give five grains of sodium salicylate. This is freely soluble in water, with a pleasant acid taste, and with a free elimination of $C O_2$ gas. This seems to form a sodium salicylate in a nascent condition, for with the dry sodium bicarbonate and dry acid when the solution is made, they combine and fulfil the idea of nascent condition.

These tablets I have been using in a four-months' hospital service in Bellevue Hospital with very satisfactory results in severe cases of rheumatism. There has been a very noticeable lack of gastric irritation even when 20 grains were given every two hours, and when the ringing in the ears was produced and the dose diminished, this ringing quickly subsided. In some patients the fever fell rapidly before the pain ceased and the pain remained for a day or two after the fever struck normal, although very much ameliorated and so insufficient as not to cause much complaint from the patient. In most patients the pain and the fever disappeared coincidentally, while in others the pain rapidly improved in a few hours, and the fever disappeared in a few hours. In two patients with a marked galloping rhythm in the heart, and with increase in the cardiac murmurs, the belief was justified that there was a beginning myocardial inflammatory process. This cardiac condition subsided rapidly in three days, and the temperature went to normal and did not afterwards rise above, even to 99 or 100. Usually, as is well-known, when the heart has become involved there is a rise to 99 or 100 for some time after the other symptoms have disappeared. In another patient with pericarditis, acute endocarditis and myocarditis and with a double rheumatic pleurisy, this form of sodium salicylate was the only form which had any beneficial influence. His pericarditis and pleurisy improved rapidly under these tablets. My supply gave out and again there was a recrudescence in the pleurisy and pericarditis. When I obtained more tablets, these processes rapidly improved under their administration. This improvement was so rapid and marked that it forced on me the impression that it was more than a coincidence.

This form of salicylates seems to enable one to get a large amount of an active salicylate into the circulation at once, in the least toxic and most effective form of the natural salicylate. The process by which this form of salicylate is made is not an economical one, and it is unfortunately doubtful if it could ever be generally commercialized. It is here reported as an exceedingly active and useful form of salicylate, with a maximum of rapidity of absorption, effectiveness after absorption, and a minimum of toxic action.

In using salicylates in rheumatism, one is impressed by the rapidity with which they act when they act well and when the form of salicylate agrees with the patient, so much so that it is my belief that if within forty-eight hours a distinctly beneficial action is not obtained with the form of salicylate which is being used for that patient, we should use some other form. If sodium sal-

icylate with large doses of sodium bicarbonate do not make a distinctly beneficial impression on the rheumatic process by the second day, it should be changed to the oleum betulae, that is, the true oil of birch, and if the patient is resistant to that, it should be changed to salophen or some other form of salicylate.

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A THORACIC ANEURISM TREATED WITH GOLD WIRE AND GALVANISM*

With Notes on a Previous Case and on Experimental Studies.

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The writer first learned of the Moore-Carradi operation from Dr. Charles L. Gibson, resulting from which he practised wiring with electrolysis four times in a previous case of aneurism, one of the innominate artery (unpublished), and also, up to the present date, in the aortas of 151 dogs, the object of the experiment work being to glean knowledge from which principles could be defined that might put the operation on a more scientific footing than heretofore. The recovery experiments still lack completion. All of the operations on the dogs were performed under ether anaesthesia. In some preliminary experimental work on this subject as well as in the recovery animals of this series, the writer received valued assistance from Dr. Robert P. Wadhams and Dr. Arthur M. Wright.

The patient here discussed was admitted to Bellevue Hospital in the service of Dr. A. A. Smith, to whom the writer is indebted for the case, and was operated upon in the service of Dr. Bryant, to whom the writer is indebted for the courtesies of his division.

The aneurism developed subsequent to the patient's having been hit on the right hip by the mud-guard of a rapidly moving taxicab and thrown onto the front of his chest and abdomen, 8 months previous to operation. His age was fifty-five; occupation, tailor.

Symptoms before Operation.—Constant pain, cough, shortness of breath and slight exertion. The pain is augmented by coughing owing to a recent spontaneous fracture across the middle of the sternum.

Physical Examination.—Pulsating expansile tumor protruding a little above the surrounding skin level, at the right edge of the sternum, having erodod through the right second costal cartilage and adjoining portion of the sternum. Crepitus felt with expansile pulsation over middle of sternum. Bruit. A little thrill on exertion.

The plan of operation, based on the animal experimentation and the lessons learned from the previous case, was, to bring as much of the introduced wire as possible into contact with the wall of the sac, and then by means of the current both to injure the areas where the wire touched, and to produce a

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fibrinous clot along the wire, which at the sites of trauma would form an adhesion with the artery during the electrical séance (Fig. 5). In dogs such adherent fibrin undergoes subsequent organization, binding the wire to the sites of trauma, while that fibrin having no opportunity for adhesion, later breaks away, leaving the wire bare. This carpentering, as it were, into the aneurismal wall of linear areas of organized fibrin, should, like ribs, fortify the structure.

The deposit of the laminated fibrin in aneurisms as a result of the wiring and electrolysis operation is ostensibly a secondary event consequent upon a

Fig. 1.

Specimen from the previous case, one of innominate aneurism, which had been wired four times. A portion of the wire (A) had been pulled out by unscientific hands. B, main mass of laminated fibrin which was attached by its base to the bottom of the recess overhanging the clavicle, whence it projected upward about 2½ inches into the cavity of the aneurism supported in the meshes of a snarl of wire (cf. Fig. 2). C, isolated loop of wire imbedded in an area of laminated fibrin about 2 inches in diameter and about ⅛ inch in thickness. D, site where three loops of wire are bound down to the intima by scar tissue. The projection here is caused by a bony prominence forming the posterior margin of a deep erosion of the clavicle. Many centrally located loops of wire presented themselves free from fibrin and untarnished. E, aneurismal opening. The probe lies in the right carotid artery, which passed across the front of the trachea. The right subclavian artery had the most distal origin of any of the vessels arising from the arch of the aorta, and passed around to the right side behind the trachea.

trauma to the intima, associated with a sluggish blood current, such as could be found in recesses of the aneurism or within a snarl of wire. In the specimen of the innominate aneurism that had been wired with electrolysis, in one situation—a recess overhanging the clavicle—where quite a number of loops of wire were grouped together in relation with the aneurismal wall, a mass of laminated fibrin had been deposited on the latter (Fig. 1, B), and projecting centrally therefrom into the cavity to a height of about two and a half inches was a further deposit of laminated fibrin supported in the meshes of a snarl of

wire (Fig. 2). Also one isolated loop of wire (Fig. 1, C) in contact with the opposite wall was associated with a deposit of laminated fibrin about an eighth of an inch thick, which covered an area of about two inches in diameter. At two other situations, loops of wire were bound firmly to the intima by scar tissue apparently (specimen disarranged by dissection) uncovered by any appreciable deposit of laminated fibrin (Figs. 1, D, and 2, F). The loops of wire examined that lay against the intima buried in the laminated fibrin, were not united to the intima by scar tissue. The sites of binding the wire to the intima by scar tissue without deposit of laminated fibrin seemed to be in regions where the blood current would have been swiftest. Many centrally located loops of wire presented themselves free from fibrin and untarnished. These

Fig. 2.

Same specimen as that in Fig. 1, showing appearance of the interior of the aneurism from the reverse side. There is seen the central projection of laminated fibrin supported in a snarl of wire, arising from the basal mass which was deposited in the bottom of the recess of the aneurismal sac overhanging the clavicle. F, isolated loop of wire bound down to the intima by scar tissue.

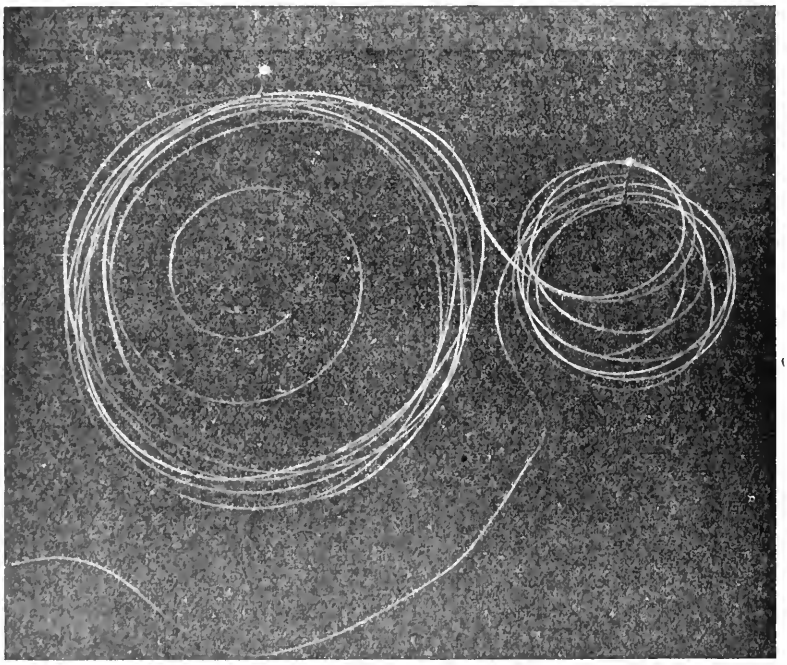
findings seemed to designate loops of wire in relation with the sac wall as the causative factor in the deposit of the peripheral laminated fibrin, and the rather closely snarled loops of wire projecting centrally therefrom, perhaps through their slowing the blood stream, as the cause of the augmentation of the fibrin deposit in an inward direction. Thus in this case the deposit of laminated fibrin over but a limited area of the sac wall, and the central projection therefrom of a mass of laminated fibrin occupying a comparatively small portion of the sac space, together with the binding of the wires to the intima by scar tissue in two places, was apparently all that had resulted from the operative procedures to give the patient considerable relief from very distressing symptoms. The

growth of this aneurism could be but temporarily arrested as a result of operation.

It is of interest that at one of the four wirings practised on this case, when a No. 26 wire coiled in loops 1⅛ to 1⅜ inches in diameter was inserted, with repeated twistings so as to snarl it centrally within the aneurismal cavity, no benefit accrued to the patient.

These findings seemed to incline one to believe that the desideratum to be attained in these cases was to get a good deposit of adherent fibrinous clot resulting from electrolysis, as well as of laminated fibrin, over as large an area as possible of the aneurismal wall, and for effecting such object it seemed

Fig. 3.



No. 28 gold platinum "Clasp" wire coiled for operation. The entering extremity of the wire was spirally shaped so that it could not go astray beyond the limits of the aneurismal cavity nor impinge directly against the sac wall. In order that the coiled loops should not get snarled before using the wire, they were bound together over a short segment of each coil by another piece of wire turned spirally around them, and the spirally shaped extremity was hooked into one of the turns. This fixation of the loops by the binding wire was in this instance done after the photograph had been taken. The spacing between any two of the longer lines on the ruler is ¼ inch. The length of the coiled wire was about 10½ feet.

proper that the wire should be gotten pretty extensively into contact with the intima so that the electric current could traumatize the latter in lines of rather close association. The principle of traumatizing the lining of the aneurismal cavity has seemed to the writer an important one, since, without it, the opportunity for the organization of fibrin is lacking.

The centrally projecting mass of laminated fibrin in the specimen suggests the possibility of producing an internal accretion to fibrin deposited peripherally within an aneurismal cavity, around a suitable nidus of wire.

If the complete obliteration of the aneurismal cavity is not produced unless enough wire be introduced centrally to slow materially the blood current, then

the operation conducted with a peripheral arrangement of the wire would be as applicable to fusiform as to sacculated aneurisms.

If that portion of the arch of the aorta proximal to the openings of the carotid arteries is the seat of operation, the danger from cerebral embolism must be considered, since the fibrin primarily deposited on the loops of wire

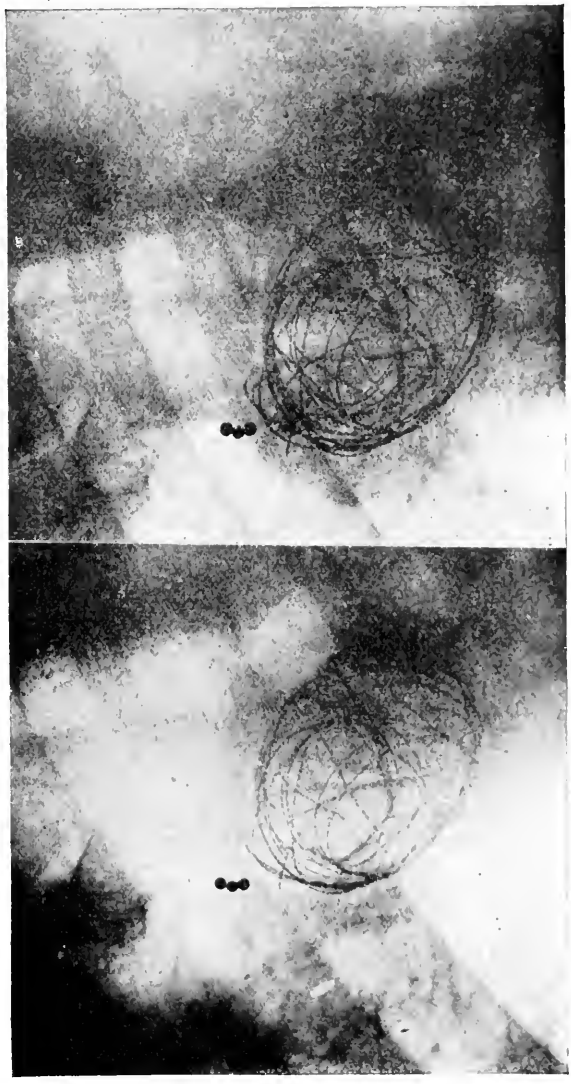


Fig. 4.

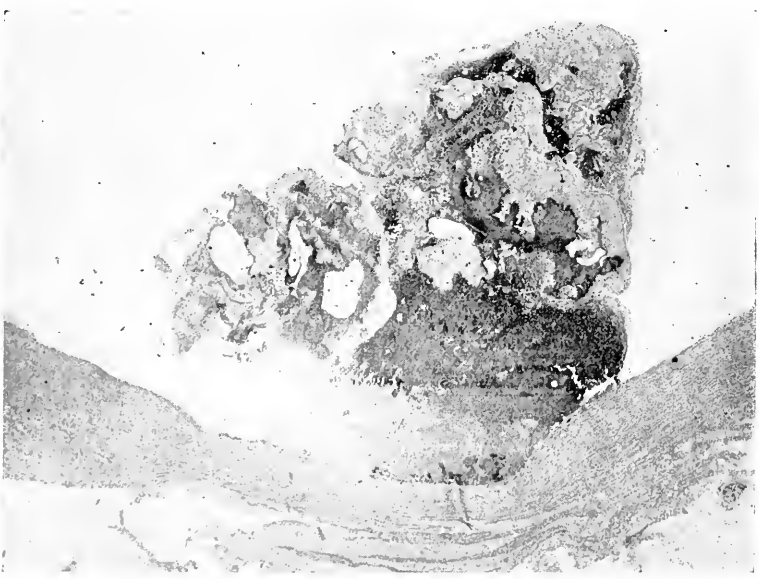
Stereoscopic X-ray photograph of the wire in situ, taken by Dr. L. T. Le Wald at the Edward N. Gibbs Memorial X-ray Laboratory, 3 months after the operation. If the picture be viewed through a stereoscope (an ordinary hand one is suitable), it will be seen that the loops of wire form essentially a globular skeleton framework which evidently outlines the limits of the sac, thus demonstrating the principle of contact of the wire with the lining of the aneurismal cavity.

not directly associated with sites of trauma of the sac lining, later breaks free, leaving these portions of the wire bare. Salinger (1) reports a case of cerebral embolism on the third day after wiring a very large aneurism of the first part of the aortic arch. Recovery ensued.

Preparation for Operation: The Wire.—The sort of wire used by the writer is manufactured in size No. 11, and is designated by the manufacturer as gold platinum "Clasp" alloy wire. A piece of the No. 11 wire of about 11¾ inches in length, when drawn down to No. 28, will produce a strand about 50 feet long. The special qualifications of this particular kind of wire which make it adaptable to the technic here advocated are (1) its resiliency, which enables it to regain its original shape after passing through a straight needle, and (2) its immunity to any solvent action of the electrolysis.

The wire used on this case, a No. 28 Brown and Sharpe gauge, was, in accordance with the proposition outlined above of bringing as much of the wire as possible into contact with the inner surface of the wall of the sac, specially coiled (Fig. 3), the first portion in 8 loops of a diameter of about 3

Fig. 5.



Shows a microscopic specimen taken from the aorta of a dog which had been killed at the termination of the wiring operation in which the electric current had been used in the strengths successively of 100 ma., 50 ma., 40 ma., 30 ma., each for 15 minutes, which demonstrates the primary agglutination of the fibrinous clot to a site of trauma of the intima. ×26.

inches and the remainder in 8 loops of a diameter of about 1¼ to 1½ inches, the idea being that the larger loops should be of sufficient size to spring against the aneurismal walls, making a good contact with the latter, and that the smaller loops, after introduction, should be snarled by twisting the external end of the wire, and then, by tilting the needle, thrown against the inner surface of the prominent protruding portion of the sac. The attempt to place the latter loops with precision, however, could not be carried out, owing to the fixity of the needle in the tissues through which it passed.

The end of the wire to be introduced was shaped in a spiral so that it could not go astray into a communicating channel, and as well that its extremity could not impinge directly against the sac wall, which might result in subsequent puncture. The latter occurrence has happened in dogs.

In order that the coiled loops should not get snarled before using the wire, they were bound together over a short segment of each coil by a wire turned spirally around them, and the spirally shaped extremity was hooked into one of the turns. This fixation of the loops by the binding wire was done after the photograph had been taken, so that it does not show in the picture. To prevent snarling during the introduction of the wire, the spiral extremity, freed from its position of fixation, should first be started through the needle before the binding wire is removed from the coil, after which the binding wire should be removed, and then the loops can be kept from crossing one another by finger pressure over the site of binding the coil, which maintains the orderly arrangement of the loops so that they will unwind without tangling. The grip for holding the coil is that between the middle, ring, and little fingers and the palm of the one hand, which leaves the thumb and index-finger free for assisting the other hand with the manipulations of the wire. As the wire thus held is now introduced, it uncoils from off the hand after the manner of uncoiling a rope. This transmits a twist through the introduced wire with the passage of each loop, which, through the resiliency of the wire, enables the loops to re-form within the sac. This technic of passing the wire through the needle should be practised in the open previous to operating. For a right-handed operator the spiral extremity must turn in the direction seen in Fig. 3. The hooking of the spiral extremity through one turn of the binding wire to prevent snarling of the wire before operation is important, since if this extremity crosses one loop of the coil the uninterrupted uncoiling of the wire will be interfered with.

The shaping of this gold platinum "Clasp" wire in coils of the desired size is very laborious, and one cannot engage to operate at short notice unless the wire is already prepared. A circle of the selected size drawn on paper can guide the shaping of the loops, and a light weight laid across the loops as each succeeding one or two are formed facilitates the progress of the work. It is not necessary that the coiled loops should lie perfectly flat. In fact, an occasional change in the direction of the curvature sufficient to cause some riding up of a loop away from the horizontal plane is of advantage, since such deviation favors a spreading apart of the loops after their introduction within the aneurism.

The needle was of gold insulated with a covering of porcelain enamel, of a calibre just enough larger than the No. 28 wire to allow the free passage of the latter without friction, which relation in sizes enables the hemorrhage through the needle to be arrested by the passage of the wire. A needle with too small a bore, through which the wire can be passed only with friction, should be guarded against on account of the liability of kinking of the wire, just as much as one with so large a bore as to permit free hemorrhage with the wire threaded through it.

Both needle and wire were boiled in a 2½ per cent. solution of sodium carbonate siccum in distilled water, but since this case was operated upon, experimental work has shown that it is better to omit the alkali.

The current, which should be direct, was taken from the illuminating fixture through a rheostat.

The operating table should be comfortably cushioned and covered with a rubber sheet.

Operation, November 24, 1911.—The needle, entering through a short skin incision, was made to puncture the aneurism near the eroded edge of the sternum, a little to one side of its most prominent portion, so as to avoid a thinned part of the sac. The three-inch diameter loops were first fed in from off the

hand after the manner of uncoiling a rope. These larger loops of the wire encountered considerable resistance from the walls of the aneurismal cavity, which seemed to show that they were of a diameter greater than that of the sac. The smaller loops were fed in like the line off a reel and then the external wire twisted twice to try to snarl them. (This technic was here in error for the accomplishment of the purpose mentioned, since, with the introduced portion of the wire fixed within the aneurism, the further feeding in of the coil by unreeling would not permit of the loops re-forming after passing through the needle.) About 10½ feet of wire were introduced. The positive pole was connected with the external end of the wire, which latter trailed over a piece of rubber dam. A felt electrode, 12 x 10 inches, backed with rubber, saturated in a 10 per cent. salt solution, to the upper margin of which, at about the level of the root of the neck, the negative pole was attached, had at the start been placed against the patient's back so as to include the area corresponding to the aneurism.

The current used on this case was determined as the result of experiments on 117 dogs. It was raised in two minutes' time to 75 ma. which was maintained for 15 minutes. Then it was reduced to 50 ma. for 15 minutes, 40 ma. for 15 minutes, and 30 ma. for 15 minutes, and then discontinued. The greatest strength of current was employed at the start, in order to cause a primary injury of the lining of the aneurismal sac at the sites of contact of the wire.

In this connection it may be stated that in the course of wiring an additional 34 aortas in dogs since operating on this case, it has been found preferable to start the current at 100 ma. and then drop to 50 ma., 40 ma., and 30 ma., each 15 minutes, with which technic the injury to the intima at the sites of contact of the wire was a little greater than when the current was raised to only 75 ma., and besides, the fibrin deposit at these sites of injury was a little greater in amount, firmer in consistence, and more strongly adherent to the arterial wall than with the weaker current. The practical superiority of the use of the stronger current would seem to be supported by the result of its application to three cases of thoracic aneurism operated upon very recently, in all of whom the diminution in the pulsation was noticeable to the patients within eight hours following the operation, which is in contrast to the observation by the patient here reported that pulsation did not markedly diminish until the tenth day.

At the end of the electrical séance on the patient, the wire was first loosened within the needle, and then the needle was withdrawn over the wire held firmly in position. There was no hemorrhage from the puncture, and the wire was pulled out a little until the resistance of the fibrin covering its inner portion was felt, and was then cut off in the bottom of the wound.

Subsequent Course.—The constant pain was relieved promptly following the operation, but on coughing the patient had considerable pain at the seat of the fracture of the sternum. Prompt diminution of pain following this operation has been noted by Hare (2).

On November 28 at 2 a. m. (fifth day) the patient had a sudden pain in his chest of great severity, which was much relieved by removal of a plaster strapping which had been placed around his fractured sternum. The tumor coincidently had become more prominent, some of which swelling, anyway, was due to oedema.

The acute pain rapidly subsided, and on the following day, November 29, he had pain only on coughing, and he coughed less than before.

December 3 (tenth day): Pulsation for the first time was found to be mark-

edly diminished. The change seemed to have come rather abruptly during the night. The patient himself had noticed it.

December 19, 1911 (twenty-fifth day): Patient has been up several days. He has no cough whatever. The tumor is little expansile. Occasional shooting pains in left nipple and inner side of the left arm. Up to this time during his stay in the hospital he has had no potassium iodide or mercury, which drugs were now administered.

February 28, 1912 (three months, 3). The patient says he can walk upstairs with perfect comfort, and that he feels perfectly well. No pain. The superficial tumor is practically obliterated, evidencing shrinkage of the sac. Pulsation can still be felt, though but very little expansile, and a bruit is still heard, showing that the sac is not obliterated. The aneurism reached its present state of quiescence about eight weeks after the operation. Very occasional dry cough.

A stereoscopic X-ray picture of the wire in situ, taken February 27, 1912, by Dr. L. T. Le Wald, at the Edward N. Gibbs Memorial X-ray Laboratory, here reproduced (Fig. 4), demonstrates the loops of wire forming essentially a globular skeleton framework, which evidently outlines the limits of the sac. Thus the principle of contact of the wire with the lining of the aneurismal cavity is seen to have been carried out in this case through the technic employed.

Principles of the Technic Here Advocated.

These principles were formulated as a result of observations on the previous case of innominate aneurism, the case here reported, and three other very recent cases, besides the experimental work on the 151 dogs.

1. The wire should be one having the properties of the gold platinum "Clasp" alloy, viz., it should be resilient and it should not dissolve under the influence of the electrolysis. The resiliency enables the loops, with proper technic, to re-form within the aneurism, so that the disposition of the wire is more or less under the control of the operator.

In contrast to this wire, a silver wire alloyed with 7½ per cent. copper (Hunner (4) and Finney (3) is not resilient, and under the influence of an electric current fluctuating between 35 and 50 ma. in the blood stream of a dog's aorta for 48 minutes it corroded, dwindling to about one-third its original size, and broke spontaneously at a point outside the artery where it lay in clotted blood. The clot deposited on this wire was fibrinous. Another wire of 12 carat gold, the alloy of which could not be determined, under the influence of a current around 50 ma. for 85 minutes, similarly corroded and broke outside the aorta of a dog, and on removal of the specimen the intra-aortal portion of the wire fractured into fragments. The clot in this case was very large in amount, dark red in color, and not at all fibrinous but of a clay-like or doughy consistence. Along the site of the wire this clot presented a metallic stain. The question would arise whether a pasty coagulum of this sort resulting from the use of a particular kind of wire would be capable of undergoing organization. This decomposition of the wire under electrolysis may elucidate happenings like those reported by Wilson (6) and by Hare (7). In the former's case the X-ray showed no trace of any wire. The wire, probably a gold one, under the influence of a current of 20 ma., had burned off during the operation, and Wilson believed it had been destroyed by the current. In Hare's case, at autopsy four months following the operation, there was no trace of 18 feet of gold wire which had been introduced into an innominate aneurism.

2. The wire should be made to come as extensively as possible into contact with the lining of the aneurism, so that the electric current can injure the same, thereby producing areas for the adhesion and organization of the fibrinous

clot deposited by the electrolysis along the contiguous portion of the wire, as well as for the deposit and organization of laminated fibrin.

In attempting to bring as much of the wire as possible into close relation with the inner surface of the aneurismal wall, the problem is a little complex because of the inability to determine definitely the diameter of the aneurismal cavity from which to gauge the size to coil the wire. The percussion note and the X-ray shadow will contribute some information. Borne out by the result in the case here reported, it would be well if the diameter of the loops of the wire as coiled before operation could a little exceed the diameter of the aneurismal cavity. If at operation this disproportion in size is found to be reversed, the diameter of the cavity being greater than that of the loops, as evidenced by the fact that the wire feeds in freely through the needle without encountering the resistance of an opposing surface, then it is the writer's proposition to give the external portion of the wire extra twists in addition to those which occur naturally from the uncoiling of the wire from off the hand, seeing to it that the former twists are made always in the same direction as the latter. This manoeuvre, through the resiliency of the wire, should throw apart the loops which have passed through the needle, snarling them into a skeleton framework of a greater diameter than the diameter of the original coil, so that the peripheral loops would then make a good contact with the walls of the aneurism.

The writer is inclined to believe that for all but very large aneurisms, the slender No. 29 filament coiled in large loops can be used to advantage. A sizable loop of this wire ought to be able to adjust itself within the confines of an aneurismal cavity of a much smaller diameter than its own, without risk of causing pressure necrosis. A No. 29 wire coiled in loops varying in diameter between 4½ and 5½ inches, on being introduced into a globular flask 3¼ inches in diameter, readily took a peripheral arrangement around the inside of the vessel. Thus this size wire thus coiled might be used for any aortic aneurism of a diameter less than that of its own loops.

3. The introduced extremity of the wire should be spirally shaped for the reasons already given (Fig. 3).

4. Unless the insulated gold needle be known to be made of an alloy which is not decomposed by electrolysis, it would be as well to test it in a dog before use on a patient.

5. In case the needle is likely to have to make a deep puncture to reach the interior of the aneurism, it can, before its introduction, be pricked through a little square of rubber dam, which will insulate its outer extremity should the latter come into contact with the superficial wound.

6. The needle and wire should be boiled in distilled water.

7. The negative electrode should be placed against the back directly over the area corresponding to the aneurism and should more than cover this area. The writer, for use on the patient, attached the negative wire at the upper edge of this electrode, but he now believes that theoretically it should rather enter at the centre of the latter just opposite the aneurism. Dr. Homer F. Swift made the suggestions that the size of the area covered by the indifferent electrode and the relation of the negative to the positive pole probably made a difference in the evenness of the clot formation along the wire in the blood stream. The experimental results confirmed the truth of the suggestions. When a small area of the animal's back was shaved for contact with the negative electrode, the clot formation along the wire was very irregular, but when the whole back from neck to hips was shaved and the animal laid on a 14-inch long electrode, the results were notably improved. It was further noticed that if the site of attachment of the negative wire to the electrode were at a low point of the

back opposite the site of entrance into the aorta of the gold wire, to which latter the positive pole was attached, the inner extremity of the gold wire lodged near the arch of the aorta would be scantily coated with fibrin, but that with the position of the electrode reversed so that the site of attachment to it of the negative wire came about opposite the arch of the aorta, then the inner extremity of the wire would receive a greater amount of fibrin deposit. These effects can be explained by the fact that the electric current passes through the body with greatest intensity in the line of the shortest distance between the two poles, and the whole length of the wire lying in the aorta is consequently acted upon evenly only when the poles are placed at its opposite ends.

8. It is as well that the external portion of the wire during the passage of the current should trail over a piece of rubber dam.

9. The principle in the use of the current as here set forth is to begin with a high current (100 ma. for 15 minutes, raised to this point at the start in 2 minutes' time), which will to a sufficient degree injure the intima at the sites of contact of the wire, and then lower the current to the strengths successively (50 ma., 40 ma., 30 ma. each for 15 minutes) which were found in the experimental work to be the combination most favorable for the production of a firm fibrin deposit that would become adherent to the sites of trauma during the passage of the current (Fig. 5). In the experiments where the 100 ma. current was used at the start, it was found that the fibrin deposit on the wire at the sites of trauma of the intima would be greater in amount than that in the intervals.

10. **The Control of Hemorrhage.**—The site of puncture should be at a thickened portion of the aneurismal wall rather than at a thinned portion. The needle should be of a calibre just enough larger than the wire to allow the free passage of the latter without friction, which relation in sizes enables the hemorrhage through the needle on its introduction to be arrested by the passage of the wire. On the withdrawal of the needle, when the puncture has been made through a thickened portion of the sac wall, the hemorrhage seems to become easily self-arrested, particularly if the wire be pulled gently outward until the fibrin-covered portion within the sac, just beyond that which had been sheathed by the needle, is felt to have been drawn firmly against the interior aspect of the puncture. In case, for any reason, on withdrawal of the needle a free hemorrhage should arise which would seem unlikely to be controlled by ordinary measures, the writer would suggest, before cutting the wire off, the starting up of the electrical current again at 50 ma. In most of the animal experiments the needle was withdrawn from the aorta immediately after passing the wire, with resulting free hemorrhage from the puncture, which, temporarily arrested by the gloved finger, could invariably be controlled by a 50 ma. current, usually within 3 minutes' time and rarely in longer than 9 minutes. In four recovery experiments where this technic was employed (the puncture being unprotected by any insulation throughout the electrical séance), and the wire was left protruding through the puncture at the end of the operation, the puncture was found at autopsy to be tight. One of these animals (highest strength of current, 50 ma.) was autopsied on the fifth day after operation, another (highest strength of current, 75 ma.) on the fourth day, and two (highest strength of current, 100 ma.) after 10 and 21 weeks respectively. In both of the latter the wires had shifted from their original positions, each having worked back through the puncture for about 2 inches into the subperitoneal tissues, where the extruded portion was found encased in scar tissue.

11. It is a well established principle that the positive pole should be attached to the gold wire and the negative pole to the electrode against the back. In a dog with the current reversed, the negative pole being attached to the wire in the aorta and the positive pole to the electrode against the back, neither trauma of the intima nor the deposit of any fibrin along the wire took place.

Sources of Trouble.

The Wire.—The right kind of wire has recently been difficult to get, until its identity with the gold platinum "Clasp" alloy was established. In order that any scientific value which his work might possess should not be nullified by a possible disappearance of this particular brand of wire from the market, the writer has had a piece of the gold platinum "Clasp" wire assayed, and it is reported to be made up of the following alloy: gold, 62.9 per cent.; silver, 17.9 per cent.; platinum, 13.4 per cent.; copper, 5.8 per cent.

The drawing of the wire to the proper fineness is difficult, as the wire is very liable to break during the procedure, and the required length may be forthcoming only after much patient work. This is particularly true in drawing the wire down to size 29.

The coiling of the wire as here suggested is laborious.

Unskilled hands may inadvertently snarl the wire, so that personal supervision of the handling of the latter is recommended.

The Needle.—The needle is very liable to get plugged by the clot which forms within it alongside the wire, which complication can take place in the following wise: If the external piece of wire over which the needle is threaded at the end of the operation be withdrawn without first cleaning the bore, it will be found that on reintroducing the wire through the needle the debris within the lumen will be jammed together into a solid mass which can be removed only by drilling. Drilling necessitates breaking of the porcelain enamel covering, and to effect re-enamelling special furnaces and special skill are required. To avoid this series of complications, the piece of wire left threaded through the needle at the end of the operation should not be withdrawn until it has first served the purpose of cleanser by being pulled alternately in opposite directions through the bore of the needle, after first soaking the latter with the wire threaded through it in cold water. A blow upon the needle, such as that received as a result of letting it drop upon a hard floor, will also chip off the enamel.

Electrical Current.—The electrical current taken from a storage battery is very liable to lose in intensity during the period of its use, and is very sure to do so if the battery has not been freshly charged. The direct current taken from an electric fixture through a rheostat gives a steady flow. With the use of the rheostat certain precautions should be taken to prevent short-circuiting, which, if it occurs, will disable the instrument. It is therefore recommended that the current be not turned on at the socket in the fixture until all the manipulations of the wires connected with the rheostat be completed. Also that when the electrical séance is finished, the connection at the socket should first be interrupted before the wires are again handled.

All the wire connections should be made tight before starting, since a loose connection might interrupt the current, which would result in giving the patient a shock. Also the main switchboard should be carefully guarded, lest, during the séance, some one should inadvertently shut the current off.

If the rheostat be used, it would be well to have an extra one in reserve.

REFERENCES.

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2. Hare: Therapeutic Gazette, 1908, p. 254.
3. May 11, 1912 (latest observation): The patient feels perfectly well, has no pain, and no shortness of breath unless he walks very fast. He walks about a mile every day. Occasional dry cough. There is a llitte increase in the force of the pulsation, which has varied in amount at different times. This change was first observed by the patient about March 6, soon after which the pulsation was observed to be of considerable violence. By April 20 there had been a slight return of symptoms, at which time the patient confessed that he had been indulging rather freely in the use of tobacco. On stopping the tobacco the symptoms promptly subsided. The aneurism has become a little prominent again.
4. Hunner: Johns Hopkins Hospital Bull., xi, 1900, p. 263.
5. Finney: Annals of Surgery, lv, 1912, p. 661.
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CHRONIC GLANDERS IN MAN.*

Report of a Case; Patient Treated with Glanders Vaccine. Apparent Cure.

WALTER C. CRAMP, M.D.

In 1906, C. D. Robin of the Royal Victoria Hospital, Canada, reported 156 cases of chronic glanders, collected from medical literature. Since this excellent review of the subject there have been reported but four definite cases, accessible to me—one by Post and one by Sieur in 1907, one by Dr. A. T. Bristow of Brooklyn, and one recently shown at the New York Surgical Society by Dr. Hitzrot. Several cases of acute glanders have occurred, however, and have been chronicled.

After reading Robin's article and noting his conclusions, drawn from an exhaustive study of his many cases, one is forcibly impressed by two salient facts: first, that chronic glanders in man is an exceedingly rare disease, unless many mistakes in diagnosis are made; second, that the mortality is extremely high, only 6 per cent. of the 156 cases reported being positively known to have resulted in a cure. These two facts alone seem to offer sufficient justification for the publication of the following case, which came under my observation in Bellevue Hospital in the service of Dr. J. D. Bryant, to whom I am greatly indebted for the privilege of studying and recording this case as one of those in which an apparent cure was obtained, the patient remaining free from the disease for a period of eighteen months. No further report of the case is possible, as the patient has returned to his native home.

In presenting this record the rule used both by Robin and Bollinger is employed to differentiate between the chronic and acute form of glanders, namely, the existence of the disease for six weeks or longer. In reporting this case, also, I have refrained from a positive statement relative to actual permanent cure, realizing that it is very difficult to state definitely just when a patient with glanders can be considered cured, as so many of those patients reported as affected, even after many months, and in a few cases after many years, finally succumb to the disease.

This patient, as stated above, had remained free from any symptoms of glanders for eighteen months, was in good health, doing hard work daily, and had also been under close observation. This patient was treated with glanders vaccine, which proved of inestimable value in controlling the disease. The vaccines were made by the New York Board of Health from a glandered horse and kindly furnished to me by Dr. W. H. Park. These vaccines were not

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standardized, and so the actual number of bacteria is not definitely determined, but estimated at fourteen to twenty million per cubic centimeter. The report of the case follows:

History.—M. M., aged 34, a Pole, a cement worker, was admitted to Bellevue Hospital, Oct. 20, 1908. There was nothing in the family history which could possibly have had any bearing on his illness. The patient's habits were good, he drank four or five glasses of beer daily, ate and slept well, and worked very hard. He was employed in making cement walks; was exposed to wet and cold a great deal, his feet often becoming wet and his clothes soaked by rain. In no way was he ever directly associated with the care or handling of horses, nor was any member of his famly. No other member of his family was ill.

Previous Illness.—One year previous to his admission to the hospital the patient had had a "stitch" in his right side. He was ill about one week and made a good recovery, except that occasionally he had a slight cough without expectoration. Generally speaking, however, he enjoyed fair health, working each day and eating well.

Present Illness.—Four weeks previous to admission, the patient's clothing became wet through exposure and the same evening he had chills and fever, followed by profuse sweating. He had similar attacks three or four times weekly previous to his admission. One week after the first chill, there appeared a swelling over the right forearm, followed the next day by one over the left forearm and right thigh, without any association of pain or tenderness over them.

Clinical Data on Admission.—When the patient entered the hospital he had a temperature of 100.2 F., pulse 120, respiration 24. Blood: Leukocytes, 11,400; polynuclears, 78 per cent.; lymphocytes, 17 per cent.; transitionals, 2 per cnt.; basophils, 2 per cent.; eosinophils, 1 per cent. Urine: amber, clear, urinous odor, sp. gr. 1.022, faint trace of albumin; many leukocytes, amorphous urates, and a few hyaline casts.

General Physical Examinations.—The patient was of medium frame and poorly nourished; mucous membranes clean, but anemic; pupils equal, reacted normally to light and accommodation; tongue heavily coated, slightly tremulous, pulse regular, of moderate tension. There were a few sibilant rales over base of the right lung; no nasal discharge nor expectoration; inguinal and axillary lymph-nodes palpable; reflexes normal.

Lesions.—Small abscess over radial side of middle third of left forearm; another on ulnar side of right forearm near elbow; another on posterior surface of calf or right leg, five inches below knee, and still another about six inches in length over posterior aspect of left thigh at the middle third. There were no signs of inflammation referable to these tumors and no pain nor tenderness associated with them. These abscesses, which were deeply situated in the muscles, were opened under cocain anesthesia, the cavities irrigated with mercuric bichlorid solution and drainage instituted. The pus was of a chocolate color, fairly thick, with no odor. The report of the findings obtained by examination of the pus is given in full below by Drs. Norris and Pappenheimer of the Pathological Department of Bellevue:

Pathologic Report.

"A deep abscess situated on the posterior and outer aspects of the middle third of the right thigh was aspirated with a sterile syringe, about 5 c.c. of pus

being withdrawn. Morphologic examination of this pus on smears and in hanging drop revealed no microorganisms.

"Cultures made on slanted and coagulated Loeffler's blood-serum, and on agar tubes, remained sterile after a week in the incubating oven. With this pus two guinea-pigs were inoculated, one subcutaneously, the other intraperitoneally.

"November 7, seventeen days after inoculation, the pig inoculated subcutaneously was found greatly emaciated, and had developed at the site of inoculation an abscess of considerable size, say 3 cm. in diameter. The abscess was incised, and the pus evacuated. Smears made from this pus revealed a few Gram-negative rods, which closely resembled in their morphology the Bacillus mallei. Besides these rods of distinctive morphology, there were found a number of involution and degeneration forms, and of globules and round masses, not staining by Gram's method, but taking on an after-strain with fuchsin.

"Cultures made from this pus yielded in forty-eight hours, on slant glycerin-agar, a growth of Gram-negative rods, resembling the glanders bacilli in morphology. With this pus, an emulsion of a glycerin-agar slant was made and inoculated into two large male guinea-pigs, each pig receiving approximately half of the agar culture emulsion.

"The pigs showed a marked and prompt reaction after inoculation, and the morning following the inoculation both pigs were very ill, with hair standing on end, extremely sensitive to the touch, especially on the peritoneum, and with greatly enlarged testicles. The pigs were found dead the following morning, approximately thirty-six hours after inoculation.

"Examination of both pigs showed a diffuse general serofibrinous peritonitis, and the typical exudate on the tunica vaginalis. The organism was found in the exudate of both guinea-pigs in smears, and by culture. The two guinea-pigs inoculated with the human pus were greatly emaciated, and were chloroformed November 13, in a moribund condition, twenty-three days after inoculation. The pig inoculated subcutaneously showed no lesions in the gross beyond the abscess at the site of inoculation in the skin. The pig inoculated intraperitoneally showed a number of well encapsulated abscesses in the peritoneum and retroperitoneal lymph-nodes, and in the skin at the site of the puncture of the needle, and abscesses of the liver.

"The bacillus which was isolated corresponded in all morphologic characteristics with that of the Bacillus mallei. It decolorized by Gram's stain. The cultural characteristics corresponded in every way to that of the glanders bacillus, giving a characteristic growth on potato and blood-serum.

"Microscopic examination of the tissues of the lesions of the two guinea pigs inoculated with human pus show the typical lesions of glanders, with numerous characteristic bacilli."

A blood-culture made also at this time proved positive.

The patient ran an irregular temperature till Nov. 5, 1908, when he insisted on returning home.

In December of the same year the patient was seen by me, he in the meantime having received treatment in one of the New York dispensaries. At that time the wounds were all healed and I decided to show him before the Bellevue Alumni Association in February, 1909, but when he was seen again, in January, 1909, two of the old wounds were discharging pus, the patient was having some elevation of temperature, chills and sweats. He was admitted to the isolation ward at Bellevue in this condition on Jan. 22, 1909, with a temperature of 102 F., pulse 108, respiration 24. He had a leukocyte count of 12,000; polynuclears, 78 per cent.; lymphocytes, 20 per cent.; basophils, 1 per cent.; eosino-

phils, 1 per cent. Staphylococci were found in smear and culture made from pus obtained from arm and leg, but no Bacilli mallei. No Bacilli mallei were found in the sputum. The agglutination test was negative.

Treatment.—January 29, 8 minims of glanders vaccine were injected into the gluteal region. In six hours the temperature rose from 100 to 104 F., pulse from 88 to 124, the patient had a severe chill and sweat, and there was considerable reaction in the region of the injection. The temperature dropped gradually to normal in three days and inflammation about the gluteal region subsided. This dose was considered too large and so on February 5, 4 minims were injected as before, the temperature rising to 102 F., and the pulse 108.

February 8, 4 minims were again injected; the temperature rose to 105.8 F., with chill and sweat. An interval of five days was then allowed before another injection of 4 minims was made. The temperature rose to 100 F. and pulse to 96.

February 21, 4 minims were injected. The temperature rose to 104.8 F., pulse to 118. About this time the patient was shown at the Alumni Association. At this time his general condition was markedly improved, but there was a slight discharge from the wound in the right forearm.

The patient insisted on going home and was subsequently treated by me at Bellevue Hospital Dispensary as follows: March 1, 1909, 4 minims of vaccine were injected. April 6, when he returned all wounds were healed and the patient's general health was excellent. He was given 4 minims, however, and again on April 20, May 4, May 18, June 4 and June 21, respectively, on which occasions there was very little, if any, reaction to the injection. Subsequently the patient was seen by me at different times, the last time being eighteen months after all his symptoms had disappeared. In October, 1910, in endeavoring to locate the patient, I was informed that he had returned to his native country, and so no further report is possible.

Conclusions.

There are several points in connection with this case and chronic glanders as a whole which I wish to bring forward.

1. Cases of chronic glanders can easily be overlooked unless one is constantly on the lookout for the disease.
2. Multiple abscesses, especially on the extremities, without definite cause, should excite suspicion, as Robin has shown that in 80 per cent. of cases multiple abscesses occur.
3. In this case there was no direct association with horses.
4. The point of entrance of the infection was unknown in this case, no abrasion on the body being present and no history of a wound being obtainable.
5. There was never any nasal discharge.
6. The patient remained apparently cured for six weeks and then showed a return of symptoms.
7. No bacilli were found in the sputum.
8. Extreme exposure to bad weather seems to be a predisposing cause.
9. This case was proved to be one of glanders by laboratory methods and by reaction to the vaccine injections.
10. This patient was apparently cured by vaccine injections.

A CONSIDERATION OF GAS BACILLUS INFECTION WITH SPECIAL REFERENCE TO TREATMENT.*

A Résumé of the Literature and a Report of Twenty-Five New Cases.

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In 1853 Maisonneuve (61) gave the first comprehensive description of the dread disease which we to-day know as gas bacillus infection, and ascribed to it the the name of "gangrene foudroyante." A few years later, 1864, Pirogoff (62) described the same disease, calling it "primary mephitic gangrene." About the same time Salleron also wrote regarding this condition, and reported 65 cases which were observed during the Crimean War, but it was not until 1871 than its infective nature was demonstrated by Bottini (63). In 1877 Pasteur (64) described a bacillus which he termed the "vibrion septique" and later Koch and Gaffky (65) found the same organism in the human body and gave it the name of "bacillus of malignant oedema."

Many cases of emphysematous gangrene were reported after this and ascribed to this etiological agent; in fact, even to-day many modern writers consider gas bacillus infection as due to the bacillus of malignant oedema in spite of the almost conclusive evidence that the infection is due to the Bacillus aërogenes capsulatus, which was first described and isolated from the blood of a human being by Welch (60), in 1891. Fraenkel (45), two years later, substantiated these findings, though working independently, and described a bacillus found in fatal phlegmons which he called Bacillus phlegmonis emphysematosae, but which proved to be the same bacillus.

It seems that in this country at least the Bacillus aërogenes capsulatus is conceded to be the specific cause of gas infections, the bacillus of malignant oedema not having been satisfactorily proven as able to produce gas in the human body, while various other organisms, at times credited as its cause, lack the necessary careful, cultural, laboratory data as proof. Welch believes that the production of gaseous gangrene in life, in the majority of cases, is due to the Bacillus aërogenes capsulatus, while Bloodgood says, "with symptoms of infection following trauma, the presence of large bacilli morphologacally like the gas bacillus, even with the absence of gas bubbles or emphysema, is practically, in the majority of cases, pathognomonic of gas bacillus infection."

Many of the cases reported, ascribed either as due to the bacillus of malignant oedema or the Bacillus aërogenes capsulatus, have the same identical picture, however, and from a purely practical, clinical stand-point it would appear that it matters little which of these is considered the etiological agent; in fact in my paper I have included ten cases reported by Dussauze (6) due to the "vibrion septique" in which the signs are identical with those ascribed to the Bacillus aërogenes capsulatus. No doubt time will prove that both conditions are the same, since many of the authentic examples of each are characterized by three cardinal features, namely, gangrene, emphysema or gas in the tissues, and a wild type of infection.

To one acquainted with the literature on this formidable disease, the subject has a bewildering effect. Its astounding complicity of synonyms is staggering, and from its birth, in its steady climb up the hill of time, it seems to have accumulated added appellations until to-day one is prone to view it with despair

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on account of its ponderous and voluminous nomenclature, there being no fewer than eleven epithets applied to it.

It is a disease which appears to be little understood and infrequently recognized except in its advanced or final stages, a disease which is so insidious in its onset, so destructive in its steady progress, and so grasping upon the claims of life that it is viewed with dread by the surgeon and as a dire calamity by the patient. Any disease which blots out five lives of every ten it attacks should certainly demand attention.

It is a disease about which to-day there seems much uncertainty and confusion. Most of the modern text-books treat it very superficially, some dismissing it with a few sentences, viewing it with extreme deference with respect to name, etiology, mortality, and treatment. One authority considers malignant oedema and Bacillus aërogenes capsulatus infection as one, while another in a 1912 edition, referring to this condition, says: "In traumatic gangrene of the extremities amputation at the highest possible point offers the only chance of saving the patient's life. Almost all the patients attacked by this infection die, and unfortunately even after amputation only 5 per cent. recover."

It is apparent from these preliminary remarks that the subject of gas bacillus infection is far from being well understood, and it seems to me that if anything, even of small import, can be added in clearing away this veil of incertitude and result in a more comprehensive understanding and elucidation of the subject, some justification at least exists for presenting these facts.

The acknowledged cause of gas bacillus infection, as suggested, in the Bacillus aërogenes capsulatus, a capsulated germ which belongs to the anaërobic group, a non-motile organism about the thickness of anthrax bacillus, with adjacent ends slightly rounded or square cut, and occurring in pairs, and at times in chains, staining by Gram's, and forming spores under certain conditions. It is normally found in the intestinal tract, having been observed in the appendix on two occasions. It has been recovered from the soil, cesspools, floor dust, and it would seem to be pretty generally distributed. There are two instances where infection has occurred in winter from injury by sleds where the ground was covered by snow at the time.

As regards the types of wounds in which this infection takes place, it may be briefly stated that it is most commonly found in wounds on which great trauma has been exercised, as compound fractures, extensive lacerated wounds with pulpification of the tissues; in grinding accidents and crushes. There are on record two cases caused by gorings, one from a goat (20), another from a deer (12); three have resulted from bites of animals, several from obstetrical cases and operations around the perineum; a few from subcutaneous injections and one from tooth extraction (10).

The mortality has been variously estimated. Welch (29) gave it as 59 per cent.; J. C. Stewart (23) at 55 per cent., while Klotz (5), in reporting 36 cases, estimated it as 50 per cent. Up to 1905 there were only 64 authentic cases on record and the general impression has been that it was a rare disease, but since then I have collected 123 others (including 10 of Dussauze (6), which were not included in the first 64, but reported 1902), bringing the grand total up to 187 cases. It is, however, a rare disease when compared to the number of injuries that actually occur, as in Bellevue Hospital during 1909 and 1911 there were 5802 cases of trauma, but only nine cases of gas bacillus infection, averaging 1 in 644.

The scope of this paper is threefold: first, to report 25 cases from Bellevue Hospital, eight of which have been treated in one definite manner, with no mortality; second, to review briefly all the cases published which are available

and thus bring the subject up to date; to classify these cases if possible; to select from the list those which have been treated conservatively and compare this group with those upon which amputation has been practised; and third, to draw deductions from the information gathered, in order if possible to secure a broad comprehensive view regarding a definite treatment of this condition.

In preparing this paper I have carefully covered the histories of 187 cases. I have not been stimulated in its preparation by any desire to prove some certain fact, so that I have not been biased or prejudiced by my findings; in fact some of the ideas which I entertained beforehand have been rudely shattered by the evidence disclosed.

The 25 cases from Bellevue are those available since 1904. For those occurring for the past eight years, and for the privilege of reporting the same I am extremely indebted to the visiting surgeons on the respective services, including Drs. Bryant, Stewart, Bissell, Hartwell, Hotchkiss, Woolsey, Smith, Lilienthal, Tilton, Gwyer, and Dennis. I have carefully excluded all those concerning which there was occasion for doubt, limiting myself to those which are typical of this infection as evidenced by characteristic signs or the actual isolation of the bacillus.

In this group of 25 cases, the Bacillus aërogenes capsulatus was found in 15 instances. In the other ten it was not sought. Twenty-four of these were confined originally to the extremities, one being the result of craniotomy, which case is interesting from the fact that in the same room in which this was performed, a gas infection had been operated upon three weeks previously, and that at the time of the craniotomy a fly lit on the wound, which fact might possibly explain the source of contamination, as Walker, of Johns Hopkins, has isolated this bacillus from floor dust. There was a gross mortality of 44 per cent., there being 11 deaths and 14 recoveries.

With reference to the type of case: 10 were compound fractures; 10 were extensive lacerated wounds or crushes; 2 were due to gun-shot wounds; 2 were post-operative, and 1 followed a burn on the lower leg about which a blanket had been wrapped. On admission this patient was pulseless, the lower leg gangrenous, the skin brawny and of many colors, and gas in tissues from toes to breasts. She died within two hours after admission, permission to operate not having been obtained until shortly before death. The interesting point in this case was the fact that although this was a typical case of gas infection, no bacilli were found, the specimen having been obtained in the upper part of the thigh far remote from the original injury, which corresponds with Lahey's (28) findings that bacilli are very numerous in the vicinity of the original wound, but not present far distant, though the tissues there are microscopically much diseased.

There were nine amputations in this group with six recoveries, or 66 2-3 per cent. Eight of the 25 cases, of which five were of a very pronounced type, were treated by incisions and either a continuous irrigation or bath, with no deaths, and it is to this group of cases that I wish particularly to call attention. Five of the eight cases were personally known to me and were treated by free incisions and peroxide continuously administered locally; another, a post-operative one in the same service, apparently an incipent case, was treated by widely opening the wound and keeping it open and freely irrigating it, while the eighth, in the service of Dr. Hartwell, was treated by a continuous bath in bichloride.

In one of my cases amputation was performed eleven days after the original injury for a mixed streptococcus infection, after the gas bacillus infection had been eliminated. Three of these eight cases were of the superficial or incipient type, the others of a severe grade and of such a malignant character that

in three instances amputation was advised and refused. The following résumé of the three will perhaps illustrate:

Case I.—Bellevue series, service of Dr. J. D. Bryant.

J. S., age twenty-six, male, was injured on December 19, 1910, in an explosion at the Grand Central Station by being struck in the lower third of the left thigh by a large, sharp rock which inflicted a long, ragged, lacerated wound of the skin and muscles and caused an indentation of the femur without fracture. Twelve hours after admission he complained of severe pain in the leg, and the temperature began to rise. The wound was dressed, but nothing abnormal found except a profuse bloody discharge. Twenty-four hours later the temperature had risen to 100.8°, pulse 136; there was emphysema in the tissues from below the knees to the costal margin, and gas was bubbling from the wound, from which also a foul odor was emanating. The muscles in the wound were greenish in color, the whole skin of the thigh of a mahogany hue and brawny to the touch. The patient himself looked septic. Amputation was refused.

Here indeed was a condition which seemed hopeless and each one who viewed it was emphatic in predicting a fatal outcome, yet under a general anaesthetic, with good, deep, free incisions and drainage, followed by a continual application of peroxide of hydrogen, this patient made a slow but good recovery. At operation the muscles were greenish in color, well upon abdomen, gas was present everywhere, and the tissues juicy. A skin graft was later done and the patient left the hospital with a useful limb. No bandaging was employed, the limb lying naked in bed, covered only by a sheet. The Bacillus aërogenes capsulatus was found.

Case II.—Bellevue series, service of Dr. J. D. Bryant.

L. L., a girl age ten years, was knocked down September 22, 1911, by a horse in such a manner as to cause a compound fracture of the right humerus with a lacerated wound one inch long at this site, as the arm came in contact with the curb. She was admitted to Bellevue and the wound dressed. She complained of pain 24 hours later and the wound was again dressed. Seventy-two hours after admission the whole arm was swollen, gas was bubbling from the original wound, crepitation was present in tissue of upper arm, and a foul odor was also evident. Temperature was 103°, pulse 104, respirations 24. Amputation was refused. Incisions were made in the upper arm, the wound freely opened, and dressed. Five hours later emphysema was extended to the chest; tissues gangrenous; there was gas both in the upper and lower arm and a second operation was performed, amputation being again refused. Free, deep incisions were made everywhere in the upper and lower arm, the axilla was opened widely and the pectoral muscles cut from their insertions to insure better exposure. The patient was afterward treated with continuous drip of peroxide for extensive necrosis of the humerus. In this case a bacillus resembling Bacillus aërogenes capsulatus was found in the smear.

Case III.—Bellevue series, service of Dr. Hotchkiss.

A. W., male, age thirty-three. Patient fell from a wagon to the pavement and was run over, sustaining a fracture of the right humerus and receiving a long, deep lacerated wound over the external and interior aspects of the right lower leg, exposing the bone. The muscles were torn extensively and much street dirt was ground into the wound. On the following day there was a profuse discharge from the wound; temperature was 102.4°, pulse 84. Two days later gas was escaping from the wound, the tissues were gangrenous, emphysema extended from ankles to costal margin, the skin was brawny and of yel-

lowish hue. Amputation was refused. The case was considered hopeless, but free incisions were made, one extending from the upper part of the thigh to the ankle, extending down to the bone on the outer side of the thigh. Peroxide was used copiously for the next 24 hours and for 10 days the wounds were saturated every two hours with it. Skin graft later. Recovery.

In this case a smear was not made. This is the third instance of an apparently hopeless case which recovered by conservative methods, and would no doubt have been classed among those in which amputation was imperative had consent been obtained. The other five cases, of the same type but less extensive, are chronicled in their proper places at the end of this paper The Bacillus aërogenes was found in four of these five; in the fifth it was not taken.

Impressed with these results, but still believing they were merely coincidences, I decided to review the whole literature, in order if possible to ascertain the experience of others. In my report I have carefully covered, as previously stated, 187 cases (1-22), including the Bellevue group, which résumé embraces all authentic cases on record known to me.

The majority of these cases were reported from this country, especially from its extreme eastern limits, but of late several cases have been reported from the West (3), which would indicate that the soil of the East is not the only habitat of this germ, as was formerly suggested.

Over 84 per cent. of these cases, taken as an entity, involved the extremities, which from a surgical stand-point were favorably located. Seventy-six were due to compound fractures; 41 were the result of extensive lacerated wounds and crushes. 21 were post-operative. 15 had their origin in gun-shot wounds; 8 were the result of subcutaneous injections of saline or from hypodermic administrations; 6 were either gynaecological or obstetrical; 6 were non-traumatic; 3 were due to bites of a lion (8), horse (6), and snake (4), respectively, all of which were of an extreme virulent character, one patient dying within 30 hours after the receipt of injury; 2 were due to gorings by animals and the remaining 5 were unclassified. In 2 cases, infection seems to have originated in the wards of the hospital, one (Bellevue group) occurring 13 days after the primary injury, the other (20) 14 days later.

For the 187 cases the gross mortality was 48 per cent. There were in all 50 amputations (barring Klotz's 36 cases concerning which no mention of treatment is made), with 18 deaths and 32 recoveries, a mortality of 30 per cent. from this form of operation. In contrast to this mortality of 36 per cent. for amputation, there were 30 cases involving the extremities, treated conservatively by incisions followed by continuous or frequent irrigations or baths, with three deaths, and all three due to complications; one from tetanus (6), one from secondary hemorrhage (6), and one of Bloodgood's (43) from a mixed infection 25 days after injury. In each case the gas infection was fully under control some days before death occurred. Nor can I find a single instance, where generous incisions were made at the outset and the wound continuously irrigated or placed in a bath, that death has taken place. Three of these 30 cases were amputated at some period in their convalescence on account of destructive effects of the disease, but not to control it.

There were nine other cases also treated in this conservative manner where the infection was located upon the trunk; all but one were superficial and all but this one, which was a deep infection in the gluteal region, recovered. Nor am I able to find here a single instance of death where the infection has been limited to the tissues external to the deep fascia, which seems extremely resistant, no matter what form of treatment was employed; two of the cases have been treated simply by incisions, the wounds having been left open. Four of these

were due to the injection of salt solution subcutaneously, the infection probably occurring from the salt employed in making the solution, as C. C. Barrows, of New York, has recently found the bacilli in the salt used in a case of an intravenous infusion followed by gas infection, which resulted fatally.

It will no doubt be argued by some that these were mild infections, local in extent and recognized early, where amputation would not have been employed anyway. But it seems this is far from the real facts, for many of these cases were similar to those I related from the Bellevue series, and of a high grade of infection, well advanced and extremely extensive, in which, in some instances, amputation had been refused and in others a more radical operative procedure was thought to prove fatal, as exemplified in the cases described by Mann (40), Bloodgood (43), Welch (4), Dussauze (6), Loving (12) and others.

There were eight cases where amputation had taken place below the limits of infection through diseased tissues or the stump reinfected, and recovery in every one resulted where the wound was opened widely, incisions made, and irrigations instituted, except in one instance complicated by tetanus.

From the foregoing facts and compilations, coupled with case histories which follow, it would seem that there are many valuable deductions to be made. In the first place it appears that when gas infection is once well established, its progress is rapid, destructive, and certain to result in death without treatment. It appears also that those cases described as mild are either those in the incipient stage, where the case is discovered early, or those cases superficial to the deep fascia.

In 15 of the 25 cases reported from Bellevue and in many of the others, pain out of all proportion to the trauma, coming on 12-36 hours after injury, was the first sign of trouble, and this was soon followed by a sudden rise in temperature. These manifestations seem to be the forerunner of this condition.

Pus is seldom present, but any wound resulting from traumatism with a gangrenous appearance, coupled with a sudden rise in temperature and pain, even without the presence of gas in the wound or emphysema in the tissues, should excite suspicion and a smear should be taken immediately, as Bloodgood (43) has reported two cases in which no gas was present but which were due to Bacillus aërogenes capsulatus.

It is very evident in reviewing the literature that few cases are recognized early, the majority being well advanced before operative means are employed, which fact is no doubt one of the reasons for its high mortality. Its incubation has been variously estimated from 24 to 48 hours, which period seems, though, to have been reckoned from the time of injury to the time the case was first seen rather than to the time of the first manifestation, and no doubt the incubation is very short, as cases have been reported coming on 8, 12, and 16 hours respectively after injury. It is also apparent that in those cases in which the disease is early recognized its progress is easily controlled by proper treatment.

In the consideration of treatment itself, it seems evident that in view of the foregoing statistics we should stop and inquire if we have not been too radical in our endeavors, and if conservatism should not rather be employed in order to avoid the baneful results and crippling deformities of an amputation. Is it not better surgery and do we not owe it to the patients to save a limb when such a low mortality is possible with less radical measures?

Prophylaxis is probably the most important aspect of treatment. All wounds on which great force has been exerted, and especially those contaminated by soil and dirt-covered objects, should be treated as if infected with gas bacilli. They should be left open where possible and thoroughly irrigated with peroxide. They should be lightly bandaged, and where possible even this should

be discarded. They should be inspected frequently and frequently irrigated, and never incased in plaster.

On the first sign or symptom of infection, a smear should be taken and examined. The wound should be freely opened if sutured, and if on the extremities, where most of these infections occur, the limb should be placed in a bath or continuously irrigated. If the condition is extensive, free incisions should be placed and the whole field widely exposed to air. No bandaging which excludes air should be employed, the limb being simply covered by a sheet or suspended in a tent.

There seems to be little reason to doubt that the causative agent being of an anaërobic variety, oxygen in some form should be employed, either in the form of air, oxygen gas, hydrogen peroxide, or water, although Lahey (28) has proven experimentally that under certain laboratory conditions the bacillus will live at least 24 hous aërobically. All forms of air have been employed with good results, but peroxide or oxygen itself seems to eliminate the infection more quickly than water. Peroxide, however, should never be employed in full strength and should never be used in confined spaces or injected directly into the tissues, as it has caused immediate death in animals when employed thus (28).

As regards amputation, which it seems should be resorted to rarely if the best results are to be obtained, spinal anaesthesia should be employed, since these patients are as a rule septic and in no condition to withstand a general anaesthetic. Even for extensive multiple incisions where anaesthesia is not present from the infection itself, it seems to me spinal anaesthesia is indicated. In one of the cases treated personally, in which amputation was resorted to nine days after the gas infection was controlled, for mixed infection, spinal anaesthesia was employed on two different occasions, and I feel positive that if the patient had been subjected to general narcosis, the result would have proved fatal.

In conclusion there are several facts which stand out prominently:

1. That the incubation is very short.
2. That the disease can be classified into superficial and deep. The former is easily combated; the latter requiring prompt and energetic action.
3. That more conservative methods should be employed in the treatment of gas bacillus infection.
4. That oxygen in some form should be used, preferably in the form of hydrogen peroxide.
5. That extreme pain coming on during the first 24 hours following a severe injury, and this accompanied by a sudden rise in temperature, may be the first symptom of gas bacillus infection.
6. That early recognition is the keynote in combating this condition.
7. That smears should be made from the original wound and not from some point distant to it.

Continuation of Reports in Bellevue Series.

Case IV.—J. O., male, admitted December 4, 1910, service of Dr. Bryant.

On the night of admission he fell down an open elevator shaft (distance unknown), striking his foot against the roof of the elevator in such a manner as to receive a compound fracture of the tibia which involved the ankle-joint. On the following day the temperature was 102°, pulse 80, respirations 20. The tissues in and about the wound were gangrenous, emitting a foul odor, and gas was bubbling from the wound, but no emphysema in the tissues. Bacillus aerogenes capsulatus found. Free incisions were made, drainage employed, and a

continuous peroxide drip begun and continued two days, followed by a chinosol bath. In 36 hours there was no gas present, odor had disappeared, but a streptococcus infection ensued. Amputation was performed eleven days after admission, below the knee. Spinal anaesthesia. Four days later, under spinal anaesthesia, amputation at thigh. Recovery.

Case V.—J. W., male, admitted November 12, 1908, service of Dr. Bryant.

On November 6 the patient struck his hand against the teeth of a second party during an altercation, inflicting a wound over the knuckle. The following day his hand became swollen and painful and was dressed by a physician. On November 12 he came to the hospital, where it was found that he had a lacerated wound over the first metacarpophalangeal articulation which was infected. Incisions were made, drainage instituted, and the hand dressed daily. On the evening of November 18 the patient complained of pain and could not sleep. The next day, on dressing the wound, gas was bubbling from the wound and the tissues were gangrenous in and about the edges of it. The temperature was 102.8°, pulse 120, respirations 28. The hand was placed in a bowl of hydrogen peroxide, and in 24 hours the condition subsided, no gas was present and the temperatur had fallen. A smear showed a capsulated bacillus.

The infection here seems to have been picked up in the ward. It was apparently in the incipient stage and recognized early.

Case VI.—Henry R., age twenty-two, admitted March 20, 1909, service of Dr. Stewart.

On the day previous to admission, he was shot by a .38-calibre pistol, the bullet entering about the middle of the right leg just external to the crest of the tibia and ranging downward and backward. The bullet was extracted by a drug-clerk who made an incision posteriorly. The patient slept on a truck all night and came to Bellevue the next afternoon, suffering from pain in the leg, with temperature at 101.5°, pulse 100, respirations 24, on which occasion there was gangrene around the margin of the wound and emphysema was present in the whole lower leg. He was shown in Dr. Stewart's clinic as a case of gas bacillus infection. Under gas anaesthesia the leg was freely incised, the primary wound enlarged. Peroxide drip was begun after wounds had been thoroughly irrigated with it. In 24 hours there was no gas in the tissues and no extension.

This was no doubt a beginning infection which was recognized early. The Bacillus aërogenes capsulatus was not sought. Recovery.

Case VII.—J. Z., age eight, admitted February 20, 1909, service of Dr. Hartwell.

Patient was knocked down by a team of horses, the rear wheel of the heavy truck to which they were attached passing over his left foot, which was extensively mangled and bled profusely. On the dorsum of the foot was a deep lacerated wound, extending from the external malleolus to the great toe; a traumatic amputation of the great toe and a compound fracture of the fourth metatarsal. On admission the foot was cleaned up under anaesthesia and a portion of the first metatarsal removed. On the following day the temperature was 102°, pulse 146, respirations 28, the patient was complaining of great pain in his foot, and was vomiting. Forty-eight hours later gas was discovered in the tissues, a foul odor was present, and the third toe gangrenous. The third toe was removed. Incisions were made on the dorsum and the original wound opened widely. The foot was placed in 1:5000 bichloride bath for 15 minutes every two hours for the following seven days Recovery. Bacillus aërogenes found.

Case VIII.—Male, age twelve. Bellevue Hospital series. Personal communication from Dr. Hotchkiss. It was a post-operative case from plating of femur following a compound fracture. Two days following the operation, gas was discovered in the tissues with a gangrenous condition in the wound from which gas bubbles emanated. The wound was opened widely, treated with peroxide. Recovery.

This was evidently an incipient case and early recognized, which subsided quickly after prompt treatment. No smear made.

The above cases are those treated by the open method plus incisions. One was amputated, but for a mixed infection.

The following comprise the remaining cases in my report which were treated otherwise:

Case IX.—C. E., boy age twelve, admitted October 8, 1906, service of Dr. Bryant.

On the day previous to admission the boy fell from a tree, a distance of 25 feet, striking the ground in such a manner as to receive a compound fracture of both bones of the forearm, which bled freely. Temperature on admission 103°, purse 120, respiration 24. On the following morning the forearm and hand were much swollen and of a bronze color. No crepitus. Chinosol bath. In the afternoon of the same day, crepitus was well marked in the forearm. Multiple invisions. By night crepitus had extended above the elbow. Arm amputation at upper third. Flaps left open. Stump irrigated with peroxide. Recovery. A large capsulated bacillus found.

Case X.—P. B., male, age twenty-six, admitted September 25, 1907. From Second Division, Bellevue.

Patient, while intoxicated, was knocked down by a trolley car, both wheels of forward truck passing over left leg, terribly mangling the foot and lower leg. Two days later, patient had severe pain, the toes were gangrenous, the tissues crepitated, and an odor of decaying flesh was apparent. Amputation below knee. Temperature 102.8°, pulse 124, respirations 28. Recovery. Bacillus aërogenes capsulatus present.

Case XI.—J. H., male, age sixty-five, admitted April 18, 1909, service of Dr. Tilton.

Patient, a driver, was knocked from his truck in such a manner that his leg was caught in the whipple tree and he was thrown to the ground, receiving a compound fracture of both bones of the leg, the fragments of bone appearing in the wound, which was a lacerated one about five inches long. The wounds were irrigated, fracture reduced. Two days later the temperature was 104°, pulse 102, respirations 22, gas present in the tissues, foul odor, amputation at thigh. Recovery. Bacillus aërogenes capsulatus found.

Case XII.—Edward C., age twenty-one, admitted June 7, 1907, service of Dr. Gwyer.

On the day of admission the patient was knocked down by a trolley car and dragged 50 feet, sustaining a large lacerated wound over the right knee, opening into the knee-joint. On the following day he complained of severe pain, and on dressing the wound gas was seen bubbling from the wound, odor foul. Temperature 102°, pulse 72. Leucocytosis 21,000, polynuclears 75 per cent. On June 14 the knee was opened by an incision across the patella. June 15 amputation through thigh. Recovery. Bacillus aërogenes capsulatus found.

Case XIII.—P. N., age thirty-two, male, admitted June 16, 1910, service of Dr. Smith.

Patient, while under influence of alcohol, caught left foot in a trolley car wheel, receiving a lacerated wound on the sole of the foot which bared the tarsal bones, also a deep wound on the dorsal surface of the foot, from which bones were protruding. Both wounds were contaminated with street dirt and bled profusely. Patient complained of severe pain 24 hours later. Forty-eight hours after admission gas was present in the tissues, gangrenous condition of wound, odor foul. Temperature was 104°, pulse 120, respirations 28. Amputation. Stormy convalescence. Recovery. Bacillus not sought.

Case XIV.—A. A., age twenty-five, male, admitted February 2, 1909, service of Dr. Hartwell.

Patient was knocked down by a car, one day previous to admission, receiving a lacerated wound about three inches long over the dorsal surface of the foot, exposing tendons and ligaments. On admission, 24 hours after accident, he was suffering severe pain. Temperature 101°, pulse 104. Three days later there was gas in the tissues around the wound, foot swollen, foul odor, tissues gangrenous. Amputation below knee. Recovery. Bacillus aërogenes capsulatus found.

Case XV.—L. V., female, age thirty, admitted September 13, 1906, service of Dr. Bryant.

Patient while cleaning windows fell from the third story, receiving a compound fracture of the right forearm and fractured tibia. Twelve hours after the accident she complained of severe pain. Forty-eight hours after injury the right arm was greatly swollen, gas in tissues of whole arm. Temperature 103°, pulse 124, respirations 40. Multiple incisions. Death. Bacillus aërogenes capsulatus found.

Case XVI.—S. N., male, age forty-two, admitted December 24, 1909, service of Dr. Walker.

Twenty-four hours previous to admission, patient's right arm was caught in a macaroni machine, sustaining a long lacerated wound anteriorly over the arm, extending from the shoulder to one inch below the elbow, and another posteriorly over the shoulder. Wounds sutured and drained. On admission he complained of pain in the arm. Temperature was 104°, pulse 120, respirations 28. Odor from the wounds foul, arm swollen, and emphysema in the upper arm and shoulder. Complained of feeling chilly. Palpation not painful. Stitches removed, dressed with formalin, bichloride, peroxide, bandage. Death. Organism not sought.

Case XVII.—F. De, male, age twenty, admitted September 18, 1911, service of Dr. Lilienthal.

Patient operated upon for glioma of brain. Four days later gas in the wound and scalp, foul odor. Death. Bacillus aërogenes found. A fly lit upon the wound during the operation, which was done in a room in which Case II had been operated upon. A possible source of contamination.

Case XVIII.—J. O., male, age forty, admitted December 31, 1909.

Patient was knocked down by a car and dragged under it. Taken to hospital unconscious, with comminuted fracture of tibia, over which was a lacerated wound, five inches long, which involved skin and muscle. Twelve hours later he complained of severe pain in leg. On the following day there was gas in the

tissues of the leg and gangrene of the foot. Temperature 104°, pulse 132, respirations 22. Amputation at thigh. Death. Bacillus not sought.

Case XIX.—A. R., male, age thirty, admitted March 4, 1906, service of Dr. Gwyer.

On the day previous to admission right leg was caught in a cog-wheel of a machine. Received a compound comminuted fracture of the tibia, several pieces of the bone protruding from the wound. On the following day the patient complained of severe pain, gas was bubbling from the wound, with a foul odor, a crepitation in tissue of the leg. Temperature 102°, pulse 120. Drainage, carbolic dressings. Amputation at lower thigh. Death. Bacillus not sought.

Case XX.—P. J., male, age forty-three, admitted September 13, 1906, service of Dr. Bryant.

On the day of admission, patient's left leg was caught between the pole of a wagon and a street car, sustaining a compound fracture of the femur, the wound in the tissues being extensive, deep, and lacerated, the biceps muscle being torn across. On the following day pain was severe, and on dressing the wound, gas in wound, tissues gangrenous, foul odor. Temperature 104°, pulse 128, respirations 32. Incisions, drainage. Death. Bacillus aërogenes present.

Case XXI.—R. S., age fifty-seven, admitted December, 1906, Second Division.

While walking along the street, patient hit in knee by a bullet which entered the joint. Twelve hours later severe pain. Thirty-six hours later, temperature 104°, pulse 118, respirations 34. Foul discharge from wound and bubbles of gas expelled. Vomiting. Death. Bacillus aërogenes capsulatus found in wound.

Case XXII.—J. B., boy, age thirteen, admitted October 20, 1906, service of Dr. Dennis.

On day of admission he was knocked down by a train and dragged along the ground for some distance, sustaining a deep lacerated wound, extending from the gluteal region to the popliteal space. On the following day crepitation in tissues about wound. Temperature 104°, pulse 160, respirations 28. Three days later crepitation to abdomen anteriorly. Death. Bacillus not sought.

Case XXIII.—P. A., male, age thirty-five, admitted April 11, 1907. Second Division.

Patient's left foot was run over by a railroad train, crushing the foot. Two days later foot was gangrenous over injured area, odor foul, crepitation in foot. Temperature 103°, pulse 120, respirations 20. Crepitation extended. Amputation of foot. Wound infected. Crepitation extended to thigh. Death. Bacillus aërogenes capsulatus found.

Case XXIV.—G. P., girl, age fourteen, admitted November 22, 1904, service of Dr. Bryant.

On day of admission she fell two stories, striking on both hands and sustaining a lacerated wound over ulnar side of left wrist, through which ulna was protruding, covered with dirt; radius also broken. Colles's fracture on opposite side. Lacerated scalp wound. Fractures reduced, drained, plaster splint. On following day severe pain, temperature 104°, pulse 110, respirations 24. Multiple incisions, wet carbolic dressing. Two days later left arm to one inch below elbow black, oedematous, with crepitation and foul odor. Extension. Death. Bacillus not sought.

Case XXV.—V. P., female, age fifty-three, admitted January 11, 1912, service of Dr. Stewart.

Three days previous to admission patient suffered a stroke of apoplexy, resulting in paralysis of right arm and leg. She complained of feeling cold in her leg, and water bottles were applied, which caused a burn midway between the ankle and knee. Around this burn a blanket had been wrapped. On the following day the leg became blue and swollen. This condition extended, and on the following day she came to the hospital with a temperature of 102°, pulse 138, respirations 42. The right foot was gangrenous, the toes white, the whole leg to groin was mottled, of a brownish-bluish tint, brawny to the touch. Crepitation was present from the ankle to breasts and seemed under great tension. A tympanitic sound could be elicited on percussion. By the time permission to operate was obtained she was pulseless and died within an hour.

Although this was a typical case, a specimen taken from the upper third of the thigh proved negative, which fact supports Lahey's experiments.

Cases Treated Without Amputation.—Loeb, 1; Mann, 1; J. C. Stewart, 1; Bloodgood, 6; Dobbin, 1; Dussauze, 9; Blake and Lahey, 4; White, 2; Loving, 2; Hotchkiss, 1; Clark, 1; Eagleton, 1; Hoseman, 1; Bellevue cases, 8.

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ACUTE PANCREATITIS WITH VERY EXTENSIVE FAT NECROSIS.*

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The occurrence of extensive fat necrosis is by no means uncommon in cases of acute pancreatitis. Although it is rarely seen in the hyperacute hemorrhage form of the disease, for the reason, perhaps, that the patient so rapidly succumbs to the intense primary shock, it is frequently observed in the somewhat milder though still acute types, where the damage to the pancreas is not so extensive and where repair of the lesion, without much impairment of function, is still possible.

Very wide variations, both in the extent and character of the fat necrosis, are found. Small, discrete, widely-scattered, pearly-colored patches, in the omental fat and elsewhere, are characteristic of the milder infections, and the severer cases are marked by massive involvement of the omentum, the subperitoneal and retroperitoneal fat, the pancreas itself and occasionally by the extension of the process to the pleural and pericardial sacs above the diaphragm.

The experimental work of Frugoni and Stradiotti (1) upon animals which had been injected with pancreatic juice obtained from a fistula established for the purpose in a dog clears many points in the histology of fat necrosis and settles some points in the mechanism of its origin and spread.

The conclusions reached by these investigators were, briefly, as follows:

1. That fat necrosis, as has been previously shown, is due to the contact of the pancreatic juice with the fatty tissue. The results of intraperitoneal injection were positive in every case.

2. The focus attacked by the pancreatic juice showed primarily a clear fat necrosis, in which later a reactive inflammation occurs which brings the process to a standstill. Newly formed giant-cells then enter the field, and finally, by the deposition of lime salts, the focus of fat necrosis is converted into a chalk-deposit.

3. A methodical chemical analysis of the fat necrosis showed the presence of a fatty acid, an earthy soap, an alkaline soap, glycerin, peptone, and tryptophane, bound together.

4. It was proven that the uninjured peritoneum exercises a strong protection over the underlying fat, preserving it from the action of the pancreatic juice, and tending to prevent the spread of the necrosis. The factor which leads to the wide dissemination of fat necrosis is most probably to be found in the existence of an accidental or physiological separation of the serous covering.

5. As it was impossible to demonstrate experimentally that fat necrosis is

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disseminated by way of the blood stream, it was thought most probable by these investigators that its spread was to be explained by regarding the lymphatics as the channels through which extension of the process occurs.

It is interesting to find the results of these experiments corresponding so closely with some of the observed clinical facts. The tendency to spontaneous cure which is evidenced clinically in some mild cases of acute pancreatitis where the diagnosis has been definitely made by an exploratory incision, is thus explained satisfactorily, on the basis of a reactive inflammation set up in the area of the fat necrosis and in the deposit of lime salts as in the spontaneous cure of tuberculosis. If it were possible to detect such cases by means of clinical diagnosis, the waiting policy would of course be clearly indicated. Unfortunately, however, the diagnosis in these acute lesions of the pancreas has not yet reached such a satisfactory stage. In the severer cases, however, with rapid onset and spread of the fat necrosis, it is obvious that the natural processes are in need of help from the surgeon, who should be able, at least in certain cases, to diminish the tension by efficient operative drainage and so relieve the condition by stopping its further spread, and, in view of the ever present danger of accidental infection from the hollow viscera in the neighborhood, to provide a means for its prevention or its possible relief.

Although it has been shown that fat necrosis is due to the liberation of a fat splitting ferment from the pancreas, either as a result of trauma, hemorrhage, or a surface exudate from the obstruction of its main duct, the reasons for its occurrence in many cases are not always clear. Obstruction of the common pancreatic duct by a stone in the ampulla of Vater forms a satisfying and demonstrable cause in some cases, especially the chronic ones, but in others no such obstruction is found.

The relation of acute alcoholism as a casual factor in cases of acute pancreatitis has been suggested by Moynihan, and in my own case, seems at least probable. By assuming the presence of a gastric or a duodenal ulcer or a catarrhal gastro-duodenitis, we have the basis of obstruction and infection which is apparently necessary to cause the final explosion. The symptomatology of acute pancreatitis is unfortunately not sufficiently characteristic to enable one to make the diagnosis with any degree of certainty, so that it comes to be made, if made at all, by means of the exploratory laparotomy, which by reason of the urgency of the conditions is often called for.

Aside from the problems of diagnosis which are of the utmost importance, the still difficult problem of appropriate treatment is constantly presenting itself.

With all this in view, and from the facts that these cases are after all among the surgical rarities, I have ventured to present to this society the clinical history of a carefully observed case of acute pancreatitis occurring in my hospital service which presented some unusual features.

Case Report.—John M., age twenty-eight, United States, driver, was admitted to Bellevue Hospital October 26, 1910. His previous history was unimportant, save that he states he had never suffered from any similar attack, and that he was a confessed alcoholic.

For two days before admission, he says he was intoxicated most of the time. On the evening before his seizure, he ate a hearty meal of a pound and a half of beefsteak, some potato salad, some bread and butter, and coffee. Two hours later he took several drinks and went to bed feeling, as he expressed it, in the best of health. He slept until 2 A. M., when he was awakened by a sharp stabbing pain in the pit of the stomach, for the relief of which he got up, went

out to a bar-room in the neighborhood, and drank two glasses of whiskey, which he almost immediately vomited. He had also a very loose movement of the bowels at this time but pain was not relieved, so he went back to bed where he continued to suffer from severe abdominal pains and vomiting until brought to the hospital in the late afternoon. His pain by this time had become more general over the whole abdomen, he had vomited repeatedly, and complained of feeling chilly and feverish and of sweating profusely at times. He was carefully examined on his admission to the medical ward, and the usual examinations of blood and urine were made. His temperature at the time of admission to the hospital was 101.2°, pulse 100, and respirations 28. He looked alcoholic, his face was flushed and he appeared very sick. His abdomen was somewhat tympanitic and tender over the region of the appendix, and especially so above the umbilicus, where the abdominal wall was rounded and very tense.

He had a leucocytosis of 26,800, with a polynuclear percentage of 89 per cent. His urine was a high specific gravity, 1034, with a trace of albumin and bile, a few hyaline and a very few granular casts and white blood-cells. His heart and lungs were negative, and his spleen was not enlarged.

On the day following, there was slight increasing resistance in the upper right abdominal quadrant and dulness in the right flank. There was also some muscular rigidity and tenderness noted over the upper segment of the right rectus. The temperature had risen to 102.8° and the pulse to 120. The case was seen at this time by a surgical colleague, who declined to operate as the condition was not clear. On the next day the temperature dropped to 100° and did not reach 102° again until three days later, the day before operation. Coarse friction developed in the left lower axilla and the patient complained of pain on deep inspiration, so that developing pneumonia was suspected. There was also some flatness on both sides just below the angle of the scapulae, and the patient appeared very sick. The blood count remained high as before and showed a polynuclear percentage ranging from 87 to 90. On October 31, five days after admission, a well-defined rounded mass was made out situated chiefly in the mid-epigastric region and reaching down to within a finger's breath of the umbilicus. The upper limits shaded off into the muscular resistance. The mass was dull on percussion, rather tender, firm, and elastic to the touch, and above it was a well-marked area of tympany. On November 1, the next day, the case was first seen and examined by the writer, and although the definite differential diagnosis of acute pancreatitis was not possible, the condition was suspected and an immediate operation advised and done as soon as preparation could be made.

Operation (Nov. 1, 1910, Dr. Hotchkiss).—Ether anaesthesia. Laparotomy above the umbilicus through the inner fibres of the right rectus muscle was done.

Upon opening the abdomen an enormously thickened omentum which was lightly adherent to the anterior abdominal wall was disclosed. This was very evidently the seat of an extensive fat necrosis which at once established the diagnosis. As the patient's condition was very bad, the gall-bladder was not searched for through the adhesions and its condition was not determined. A finger was thrust through the lesser omentum, which was very thick, and the seat of extensive fat necrosis, and the lesser peritoneal cavity was opened, giving vent to a considerable quantity of bloody fluid under pressure together with numerous lumps of tissue, apparently necrotic fat and fibrin. The pancreas was easily palpated but its exact condition could not be made out any further than to discover that it felt harder in some places than in others. A large cigarette together with a wrapped split drainage tube was carried to the bottom of the

lesser sac, the abdominal wound closed to the drains, and the patient returned to bed.

His condition improved somewhat after the operation, the pulse becoming a little less frequent and the temperature tending generally below 101°. The blood count however continued high, appetite failed, and the patient grew steadily weaker and much emaciated. It soon became evident that the interior drainage alone was inefficient, and a second operation was advised and done on November 19. At this operation, the anterior sinus was excised and the wound reopened. The patient was turned over on his face, and a broad incision parallel with the twelfth left rib was made, opening into the lesser sac from behind. From the large pieces of necrotic tissue which were washed away it was evident that we had to deal with a very extensive fat necrosis of the retroperitoneal tissues. A large fragment of tissue which was washed from the wound was submitted to the pathologist and found to be a portion of necrotic pancreas. This piece and others resembling it were dry, crumbly, and evidently in a condition of fat necrosis. Through-and-through tube drainage was arranged and the patient who was in considerable shock was returned to bed, where he quickly rallied. The temperature fell but rapidly rose to 104° on the second day and then gradually receded. The drainage was very profuse for the first few days, necessitating frequent change of dressings, and irrigation continued to bring away large amounts of necrosed fatty tissue and purulent fluid.

The discharge soon diminished and the drainage openings contracted rapidly and soon were nearly closed. The patient remained very white, feeble, and emaciated, and all our resources were taxed to provide him with food which could be assimilated. For a time he barely held his own and then his appetite began to return and he began to gain in strength and flesh and color. On December 16, a faintly positive Cammidge reaction was obtained, and the posterior wound had nearly closed. The rubber drainage tubes were removed as early as possible for fear of pressure necrosis. The temperature fell to nearly normal, the pulse had much improved in quality, and the anaemia was less apparent. On December 21, however, just as his improvement had become well established and he was eating well and enjoying his food, he had a sudden profuse discharge of fluid from the anterior sinus, which was found to contain most of his recent meal. The diagnosis of stomach perforation was confirmed by allowing the patient to drink water tinged with methylene blue, which almost immeditely flowed out of the wound sinus. The patient's strength began to fail very rapidly and it became evident if he were to be rescued from starvation an attempt would have to be made to find and close the opening in the stomach.

Accordingly six days later, on December 27, the patient was taken to the operating room for the third time and his abdomen again opened under ether anaesthesia. As the attempt was extremely hazardous in his very weak condition, he was first infused with saline solution. His abdomen was painted with tincture of iodine and the track of the original anterior incision together with the sinus was excised. On exploring the anterior wall of the stomach no perforation could be seen and as the organ was adherent lightly along the greater curvature, and as much handling of the viscera was out of the question in the patient's feeble condition, an incision was rapidly made through the anterior wall of the stomach and a small perforation quickly discovered upon the posterior wall, just above the greater curvature. The adhesions along the greater curvature were easily broken up and with one finger in the cavity of the stomach the perforation was easily pushed forward into the wound, where it was quickly surrounded with a purse-string suture, dropped back, and the incision in the stomach closed, with the loss of but little time. The abdominal wound was closed

to a small cigarette drain and the patient put back to bed in a condition of severe shock, from which, however, he quickly rallied. A period of careful rectal feeding followed and after a week, careful stomach feeding was resumed. The patient gained very rapidly in flesh and strength and went on to an uncomplicated and apparently perfect recovery. He reported several months after his discharge from the hospital, showing an enormous gain in weight and being apparently in perfect health.

The question which naturally arises with respect to the late perforation of the stomach, as to whether it was due to pressure necrosis or to pancreatic digestion of a damaged portion of the stomach wall, or to the pre-existence of a gastric ulcer, can of course not be answered.

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SARCOMA OF THE SMALL INTESTINE.*

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Malignant growths of the large intestine are unfortunately comparatively common, carcinoma being the type of growth in the vast majority of cases. Mayo (1), in a report of 100 resections of the large intestine, 61 for malignancy, only reporting one case of sarcoma. In the small intestine malignant growths are less frequent, and sarcoma in its various forms is by far the most usual type of growth. Kanzler (2) in 1906 could only find 23 published cases of carcinoma of the jejunum or ileum, to which he added two of his own. Primary sarcoma of the small intestine, however, is sufficiently unusual to make the following case worthy of report.

M. C., male, age twenty, native of Turkey. His family and previous history were negative. Six weeks before his admission to Bellevue Hospital on July 1, 1911, he noticed a mass in the lower part of the abdomen. It gave him an uncomfortable feeling when he was up and about, but the pain disappeared on lying down. Tumor grew larger and pain became worse, and he was obliged to stop work three days before admission to the hospital, because of weakness and discomfort. No urinary symptoms.

Physical examination showed a well-developed, anaemic looking but not emaciated patient. The abdomen was somewhat distended but not rigid, and there was no evidence of free fluid. In the median line and slightly to the right of it, midway between the umbilicus and the symphysis pubis, could be felt a firm mass about the size of the fist. It was somewhat irregular in outline and very slightly movable.

A median abdominal incision 12 cm. long was made over the mass, and on opening the peritoneum, an irregular white tumor involving a coil of the ileum and the glands in the mesentery of the intestine at this situation was demonstrated. The tumor was adherent to the omentum and slightly to the peritoneal coat of the bladder, and in attempting to free and deliver the mass, the lumen of the intestine, the wall of which was apparently replaced by tumor tissue, was broken into. The tumor with at least 5 cm. of normal intestine on each side of the growth, in all about 45 cm., was resected and a lateral anastomosis per-

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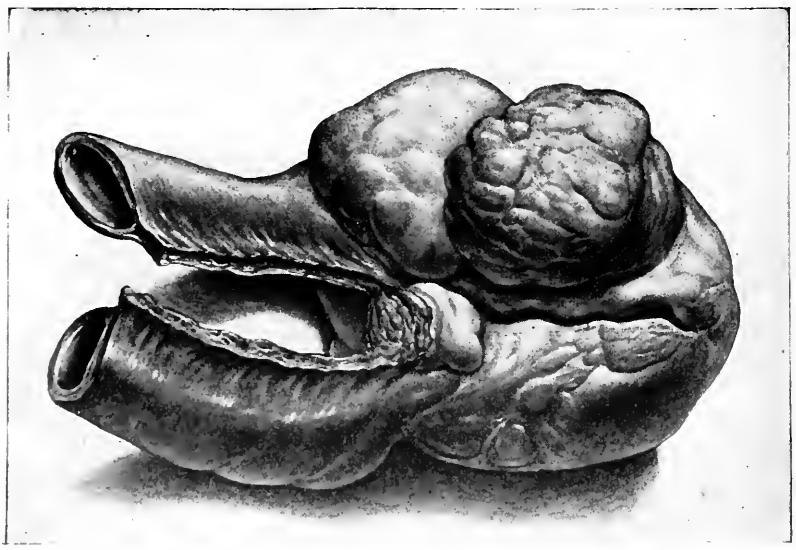
formed. The wound was closed without drainage. The bowels moved without catharsis or enemata the day following operation, and except for a slight infection in the lower angle of the wound, due probably to contamination when the intestine was broken into, convalescence was uneventful. In the three months following the operation the patient had gained 22 pounds in weight. The pathological report showed lymphosarcoma.

The most complete report and analysis of cases of sarcoma of the small intestine are those of Moynihan (2), who gives brief reports of 40 cases submitted to operation up to 1906; and of Lecène (4) (1907), whose article is the most exhaustive on the subject, and includes an analysis of 89 cases, both operative and non-operative. Since these statistics have been assembled, additional cases have been reported by Barling (5), McGlinn (6), Erdmann (7), Anderson (8), Stern (9), and Goebel (10), each one case, while Bondareff (11) reports two cases, LeRoy (12) reports three cases, one a case in which resection was per-



Sarcoma of small intestine, showing specimen removed at operation.

formed, one case found at autopsy, and one occurring in a tuberculous woman. Munk (13) reports six cases of sarcoma of the small intestine and one of the splenic flexure from the Breslau clinic between 1900 and 1908. In addition to these cases reported as sarcoma Fletcher (14) reports a case of primary lymphadenoma and Scudder (15) one of malignant lymphoma or lymphosarcoma of the small intestine for which resections were performed.

Pathology—Round-cell sarcoma occurs in about half the total number of cases reported, spindle-cell and lymphosarcoma, are the next most frequent varieties, while myosarcoma, alveolar sarcoma, fibrosarcoma, giant-cell sarcoma, myxosarcoma, and angiosarcoma are also reported. Lecène divides the cases into two kinds, circumscribed and diffuse, single tumors occurring twice as frequently as multiple growths. Ulceration occurs in over half the cases, but cicatricial contraction, such as occurs in carcinoma of the intestine, causing stenosis and obstruction, is very unusual. The ileum is the most frequent site of

the tumor, the jejunum, ileocaecal region, and junction of the duodenum and jejunum being involved in the frequency of the order given. The mesenteric glands are involved early in the course of the disease. Perforation of the intestine may occur.

Symptoms.—The first symptom noticed is usually the presence of the tumor in the abdomen or abdominal pains or discomfort. There may be some abdominal distention, but complete obstruction is rare. When obstruction occurs it is usually due to growths in the mesentery or to kinks or adhesions of the intestine. A small tumor may be the cause of an intussusception, which latter condition is not infrequently the first symptom of the growth. Anaemia and cachexia appear quite early, the latter being the usual cause of death. Ascites or melaena is rare. There may be some elvation of temperature but usually not.

Prognosis.—Of course without operation the prognosis is fatal. Lecène gives as the duration of illness four to six months in round-cell and lymphosarcoma; and eight to ten months in spindle-cell sarcoma. The primary mortality from operation is high—57 per cent. In Moynihan's series of 40 cases, five deaths being due to recurrence. The recorded cases since these statistics would lower this mortality. Bondareff states that recurrence occurs in 95 per cent. of the cases of round-cell sarcoma, although he reports one case without recurrence for three years, and Steinthal (16) one free for three and a half years, and a case of spindle-cell sarcoma free for four years after operation.

Statistics, however, as to operative mortality or infrequency of recurrence are of little value, as so many elements, such as size, distribution, situation, duration, glandular involvement, and involvement of neighboring structures, are the determining factors.

Treatment.—If the diagnosis is made early enough to completely remove the growth there is some prospect of a cure, but even if there is prospect of recurrence, the symptoms may be relieved and life prolonged by as complete a resection as possible, well clear of the involved intestine, with the removal of the mesentery and glands, followed by an anastomosis either by the end-to-end or lateral method.

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STRANGULATED FEMORAL HERNIA.*

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Strangulated femoral hernia is a surgical condition in which early diagnosis and prompt operation result in a fairly low mortality; while failure to recognize the condition and consequent delay directly increase the danger and operative risk. The fact that a large proportion of these cases are not diagnosed promptly, or an incorrect diagnosis is made with a resulting delay of the necessary surgical treatment prompts the writing of this paper, with the brief report of the following nine cases:

Case 1.—R. W., woman, aged 54, had swelling in groin for two years, which had increased in size for past six weeks; vomiting four days before she was referred to Bellevue Hospital. Pain in swelling was complained of for only twenty-four hours before admission. Intestine was found blue and congested, but circulation returned and hernia was reduced and ring sutured. Patient was discharged as cured on twenty-ninth day.

Case 2.—S. F., woman, aged 28, had swelling the size of a marble in groin for three years; had sharp pain in abdomen after lifting weight, four days before admission to Bellevue Hospital. Pain was accompanied by persistent vomiting and absolute constipation. On admission, gangrene with perforation of the strangulated intestine had been present a sufficient time to result in suppuration in the sac, and the formation of a mass resembling a broken-down bubo. General peritonitis was also present. The gangrenous intestine was resected and united by a Murphy button. Median abdominal incision was made to sponge and drain peritoneal cavity. Button was passed on twelfth day. Patient was discharged as cured on thirty-fifth day.

Case 3.—Patient M. T., woman, aged 63. Strangulation was present for two days; treated as an enlarged gland and gastritis until fecal vomiting appeared. Operation in private house. Color returned to middle of loop of strangulated intestine, but a band existed at both ends of the loop, where pressure on the sharp edge of Gimbernat's ligament had thinned out the intestinal wall to almost resemble tissue paper. Six inches of intestine were resected and united by a Murphy button, which was passed on the sixteenth day. Recovery followed.

Case 4.—L. B. ,woman, aged 42, two days before admission to Bellevue Hospital had sharp pain in lower abdomen, followed by persistent vomiting, which became fecal; absolute constipation. Patient did not know she had a hernia; was sent into the hospital with a diagnosis of acute gastritis. Intestine was in bad condition; did not react. Resection of 2½ inches was performed and end-to-end anastomosis by suture through medium abdominal incision. Bowels moved four times on third day after operation, and continued to move daily until seventh day. Temperature was high the day following operation and continued high until death on the seventh day. Post-mortem examination showed no leakage or obstruction at line of anastomosis, but an evidently virulent peritonitis with very little exudate.

Case 5.—M. W., woman, aged 62, had swelling in groin for twenty-two years. Three days before admission to Bellevue Hospital, after eating corned beef and cabbage, she began to vomit and have abdominal pain; vomited for seventy-two

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hours. After forty-eight hours, vomitus became brown. Absolute constipation was present for five days. Operation showed strangulated small intestine in which the vitality had not been lost. Intestine was reduced and ring sutured. Patient was discharged as cured on twenty-first day.

Case 6.—L. S., woman, aged 60, had swelling in groin for one year, which was treated as enlarged gland with ichthyol. Vomiting and pain were present weny-four hours before admission to St. Luke's Hospital. Operation showed strangulated intestine in which the color returned to the middle of the intestinal loop, but the portions where the sharp edge of Gimbernat's ligament had caused pressure and constriction were sufficiently damaged to require resection. Resection of 5 inches was done through median abdominal incision; end-to-end anastomosis by suture. Patient was discharged as cured on nineteenth day.

Case 7.—E. G., woman, aged 68, had hernia for fourteen years. Four days before admission to Bellevue Hospital she became nauseated and had abdominal pain. Hernia became large and irreducible and painful. Vomiting continued and became fecal. Absolute constipation existed three days before admission; patient in poor condition. Operation, under local anesthesia was rendered difficult by a partial ankylosis in semi-flexion of the hip, due to rheumatoid arthritis. Intestine was not damaged, and returned. Patient was discharged as cured on thirty-fifth day.

Case 8.—M. B., woman, aged 41, had swelling in groin one month. Two days before admission to Bellevue Hospital, she was seized with sharp abdominal pain, and vomiting which became fecal. Mass became harder and more painful. Abdomen became distended and constipation was absolute. Operation showed a strangulated femoral hernia of the Richter type, in which the constricted portion had almost ulcerated through. Resection of 4 inches was done, with end-to-end anastomosis by suture. Patient was discharged as cured on twenty-eight day.

Case 9.—B. S., woman, aged 35, had had hernia for three years. It had always been reducible up to three days before admission to St. Luke's Hospital. There was no vomiting; bowels had not moved for two days; there was slight elevation of temperature. Flatness over mass; no impulse. Operation showed strangulated omemtum, in sac very tense with fluid. Omentum was resected; ring closed. Patient was discharged as cured on fourteenth day.

The brief report of these nine cases shows the frequency of delay (two to four days) before operation was performed, and the errors of diagnosis and failure to recognize the early necessity of surgical interference. Five of them required resection of the intestine. One patient died; of the four not requiring intestinal resection, all recovered. The damage to the intestine, however, does not depend alone on the length of time constriction has been present but also, to a large extent, on the nature of the constriction—the larger hernias seeming to be less apt to be damaged than those with a small ring where a small knuckle of intestine, or even a Richter's hernia, is tightly constricted by the sharp edge of Gimbernat's ligament.

Diagnosis.

Femoral hernia occurs much more frequently in women than in men. The nine patients referred to above were all of the female sex. Of fifty-nine patients suffering from strangulated femoral hernia, from the records of St. Luke's and Bellevue Hospital, only seven were men—a proportion of less than one in eight.

When the hernia is reducible, the diagnosis between the femoral and inguinal

variety is made by the direction in which the sac contents return to the abdominal cavity, and the relations of the sac, and particularly of the neck of the sac, to Poupart's ligament and the pelvic spine. When strangulated, the differential diagnosis between femoral and inguinal hernia must be made by the relations or the tumor to the two anatomic landmarks above mentioned. One of the most frequent errors in diagnosis is the mistaking of a small strangulated femoral hernia for an enlarged or broken-down femoral gland. The impulse on coughing is lost and the percussion note when the sac fills with fluid is no longer tympanitic. When perforation and gangrene of the intestine occur, the signs of suppuration appear in the mass.

The above are the local visible signs. But in a number of cases strangulation of a femoral hernia occurs, in which the patient has not been aware of the previous existence of a hernia, and in which the general abdominal symptoms are so marked that the patient does not complain of the local condition, and unless the attending physician makes a careful physical examination, a diagnosis of acute gastritis or intestinal obstruction of unknown cause may be made. The case histories cited show that the first symptoms of strangulation are frequently general abdominal pain and vomiting, and even with a superficial examination, a small strangulated femoral hernia, especially in a fat woman, may escape detection. Hence, in every case of severe general abdominal pain with intractible vomiting, or when signs of intestinal obstruction are present, the femoral rings should be carefully examined.

Treatment.

The facts that femoral hernia is especially liable to strangulation, and that it is with the greatest difficulty kept reduced and almost never cured by means of a truss, while operative treatment by practically any of the many different methods of operation for radical cure is nearly universally successful, indicate the wisdom of the advice of operation when the diagnosis of femoral hernia is made. Obviously, sufficiently early diagnosis and operation in the event of strangulation would eliminate the necessity of resection, and therefore greatly lessen the mortality.

When operating in the presence of strangulation, if after the relief of the constriction, usually by nicking or dividing Gimbernat's ligament, the color of the strangulated intestine returns to normal, showing that the intestine is viable, it should be returned to the abdominal cavity and the crural ring closed. If, after waiting, and the application of warm towels, the color does not return to normal, and the intestine or part thereof is obviously damaged, one of the several procedures may be done. In certain cases, the return of the circulation in the loop of the intestine is prevented by traction on the mesentery, in which event the intestine may be partly or entirely returned temporarily to the abdominal cavity for a short time until its viability can definitely be determined. The latter suggestion has recently been made by Plummer, (1) who writes that he could find no mention of it in the text-books. Surely the value of this suggestion is so obvious that it must have been used by other surgeons when it was evident that it was traction on the mesentery which prevented the return of circulation to the intestine. In the presence of any evidently gangrenous area on the intestine, or infection of the sac contents, the temporary return of the intestine risks infection of the general peritoneal cavity and, of course, should not be done.

It has been suggested that after relief of the constriction, if there is still question of the viability of the intestine, it may be left in place surrounded by gauze, wrung out in hot salt solution, until this question is determined, when it

may be replaced and the hernia repaired or a resection done, as indicated, at a second operation (operation in two stages); or the doubtful intestine may be returned just within the ring and held near the opening by means of sutures, the hernial ring not being closed. These procedures are certainly preferable to returning damaged intestine to the free peritoneal cavity, but if a secondary resection should be necessary, the chances of recovery of the patient will probably not have been increased by the delay, and it would seem wiser to resect at once in doubtful cases.

It has also been suggested that in those cases in which the circulation returns to the loop of the intestine, except in a band where the pressure of Gimbernat's ligament has caused what is practically a pressure necrosis, as in Cases 3 and 4, the damaged area might be inverted without resection. Hodge (2) reports a case of intestinal obstruction following this operation, which necessitated a subsequent resection ninety-six hours later. This would seem a not unlikely result where any considerable breadth of intestine had to be inverted. Statistics also show the very high mortality (90 per cent. in the combined Bellevue and St. Luke's statistics) of cases in which an enterostomy has been done, although this is probably used only when the patient's condition has been allowed to become so desperate that a resection and anastomosis would almost certainly be fatal.

When the intestine is gangrenous or obviously damaged beyond viability, resection and anastomosis should be performed. Whether this can best be accomplished through the enlarged femoral opening or through a secondary abdominal incision will depend on the size of the enlarged femoral opening, and also on the length of the mesentery of the loop to be resected. If the mesentery is not sufficiently long, traction on the loop of the intestine interferes with its circulation, and in such cases an abdominal incision allows greater freedom in performing the anastomosis, and better judgment in determining the amount to resect, doing less damage to the intestine on each side of the anastomosis. Also, there is sometimes difficulty in returning the anastomosed intestine through a not greatly enlarged femoral ring. On the other hand, the performance of the anastomosis through a secondary abdominal incision necessitates the reduction of the damaged and perhaps gangrenous and infected intestine, and the carrying of it across a free, clean area of the peritoneal cavity to the second incision, with the danger of peritoneal infection. As to the method of anastomosis, whether lateral, end-to-end, by suture or Murphy button, depends on the speed, skill, familiarity with the operation, and personal preference of the surgeon. Lateral anastomosis can usually be performed more rapidly and is generally believed to be safer and easier than an end-to-end anastomosis, and is probably preferably done when operating through the secondary abdominal incision. It requires a longer loop of intestine, however, than it may be possible to pull down through the femoral ring in the event of a short mesentery, and in such cases an end-to-end anastomosis is preferable. Also, it is more difficult, or may be impossible, to return into the abdominal cavity without very greatly enlarging the femoral ring.

The method of closing the ring after tying off and displacing the neck of the sac, employed in the nine cases reported, was by means of a purse-string suture through Poupart's and Gimbernat's ligaments, the pectineus sheath and muscle, and the sheath of the femoral vessels. This has been found most satisfactory in these cases, as well as a number of non-strangulated femoral hernias. Sprengel, however, has collected fifty different methods of repair of femoral hernia, and Ochsner states that a cure will be effected by carefully tying off the

sac alone. Recurrences, however, have occurred where no attempt has been made to unite the muscular and ligamentous strictures forming the crural ring, and is especially apt to occur where enlargement of the ring has been necessary to relieve constriction in a strangulated hernia.

Finally, as to the anesthetic: Relief of the constriction, reduction of the hernia, and repair of the ring, and even resection and anastomosis, can be obtained under morphin and local anesthesia, with very little pain in most cases, and in the presence of fecal vomiting, even after lavage, this is a safer, though slower method of procedure. However, in cases in which it is necessary to make considerable traction on the mesentery in order to resect, the pain is severe and a general anesthetic necessary.

Conclusions.

1. Because of the danger of strangulation and inability to cure by other than operative measures, operation should be advised when a diagnosis of femoral hernia is made.

2. Sufficiently early diagnosis and operation would prevent the necessity of intestinal resection, and thus lessen the mortality.

3. Intractable vomiting with pain, either abdominal or localized in the groin, especially in women, should indicate careful examination of the femoral rings, even before it is obvious that intestinal obstruction exists.

4. Operations should be performed as soon as the diagnosis of strangulated femoral hernia is made, if gentle attempts at reduction fail.

5. If the strangulated intestine is damaged beyond viability, resection and anastomosis should be performed. When there is a sufficiently long mesentery not to hamper the operation, this may be done through the primary incision made over the femoral ring; otherwise, a secondary abdominal incision should be made.

6. Except in the most desperate cases, when the patient's condition is so extremely bad that it is impossible to perform an anastomosis, an enterostomy, even as a temporary resort, should not be done.

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ENTEROLITHS WITH A REPORT OF A CASE.

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In the herbivora, and in animals which have the habit of licking their coats as well as the coats of their fellows, intestinal calculi or enteroliths are not uncommon, and frequently attain considerable size. They are composed of husks or matted hair, with a variable amount of hard fecal matter included.

Oriental "Bezoars" (1) are intestinal concretions, which probably were formed in "Capra Aegarus" and Antelope dorias. They are usually spherical or oval, of a dark, olive green color, due to biliverdin, with a smooth, shining exterior, and are composed almost entirely of lithofellic acid.

False "Bezoars" which come from the eastern countries, have a brownish black color, and are composed chiefly of ellagic acid, probably derived from tannin contained in the food consumed by animals which yield them.

In the human intestinal tract many foreign substances find their way, but it is rare for any of them to form a nucleus of a concretion of sufficient size to

cause intentinal obstruction, unless perchance it lodges in Meckel's Diverticulum, or in some pouch of the intestines, as in the case reported by D. M. Grieg, where the obstruction was relieved by the spontaneous evacuation through an umbilical fistula of an enterolith, 2½ inches in circumference and weighing 24 gms. This may have been a true enterolith, or a phosphatized gall stone, which is a frequent cause of intestinal calculi in man. Gall stones may be of such size or of sufficient quantity as to give symptoms of obstruction.

U. Gabbi (2) reports such a case in a peasant, 45 years of age, who, one and one half years after an attack of hepatic colic, was suddenly seized with a violent pain in the right inguinal region, followed by nausea and vomiting. The pain became rapidly more severe and generalized. The abdomen greatly distended, accompanied by tenesmus without evacuation. Temperature only slightly elevated and pulse accelerated. Purgatives and enema relieved the distention sufficiently to disclose, in the region of the ileo-caecal valve, a palpable, freely movable tumor with a hard nodular surface. Later, a hemorrhagic exudation occurred in the peritoneal and pleural cavities. This was removed by aspiration. Stools became normal, urine contained no albumin or sugar, but much urobilin. Patient began to recover and left the hospital but, according to his story, entire restoration to health came only after a chestnut sized, whitish stone had been evacuated in a stool, followed by eight bean sized stones and about fifty small stones.

According to Gabbi, the large number and small size of most of the evacuated stones exclude a diagnosis of enterolith of the feces. Since an attack of hepatic colic had previously occurred, it is probably that these gall stones had remained in the intestines for a year and a half and only after that period had given rise to the above symptoms. It is a fact that gall stones, during a protracted stay in the intestines, become phosphoratized and adopt the characteristics of true enteroliths. It is also a fact that gall stones may more or less completely close the intestine, and thus produce attacks of severe intestinal colic, especially if they lodge in large numbers in the narrow portion of the ileum, just above the ileo-caecal valve. Gabbi attributes the hemorrhagic quality of exudate to previous malarial attacks.

There are also found in the intestines, and sometimes in the stools, small concretions, whose central mass consists of undigested food residue or inspissated mucus, while the bulk of the material is composed of ammonium magnesium phosphate or perhaps magnesium phosphate; but many may include calcium phosphate or carbonate and occasionally calcium and magnesium soaps and albuminous matter.

E. Doetz (3) and C. H. Bedford (4) report cases of intestinal sand, but careful investigation failed to reveal the character or the origin.

The case reported below presents several interesting features, i.e., the size and character of the enterolith, the symptom complex leading to a mistaken diagnosis, the age and mental condition of the patient and her subsequent history.

Case.—D. K., age 17 years, single, native of Russia, by occupation a shirt waist maker, was admitted to the service of Dr. Taylor at Fordham Hospital, on July 12th, 1912. Nothing could be learned of her family history. Her parents were of the very poor peasant class of Russia. She recalled no disease of childhood, but had always been anaemic and weak with poor appetite and constipated bowels. Menstruation began one year ago, her periods being irregular in duration and occurrence and accompanied by severe pain. Patient lives in very poor hygienic and moral environment.

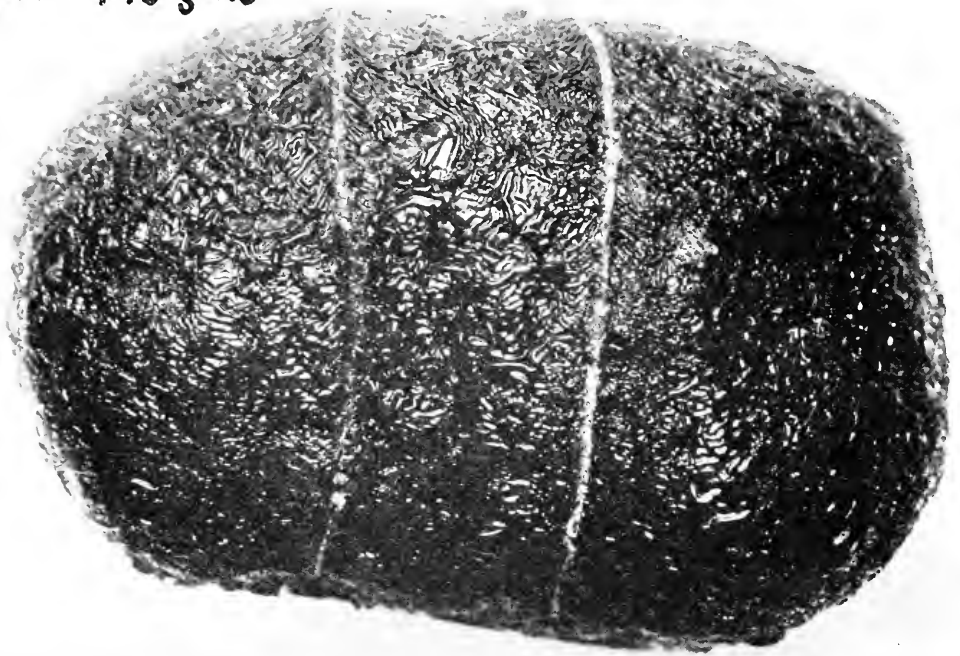
At the age of ten, she first noticed a swelling of abdomen in the hypoga-

Measurements - 15.4 x 12.0 x 10 cm.

Circumference - Long. - 43 cm.

" Short 34.4 cm.

Weight - 945 gms



tric region. This tumor has gradually increased in size, has caused no pain but, at intervals has been slightly tender. Up to four months ago, she had suffered from severe constipation; but since that time she has had large and copious movements. Her menstruation ceased entirely four months ago. She has had no vaginal discharge or uterine pain. Denies sexual intercourse. There has been no nausea or vomiting, no chills or fever. She has not lost flesh or strength.

Physical Examination.

Very well nourished girl, noticeably lethargic, slow and indefinite in answering questions. Complexion is sallow, pupils equal and react to light and accommodation. Tongue is moist, white and heavily coated. Heavy pigmentation of areolae about nipples. Nipples not erectile, no secretion expressible. Breast flabby.

Examination of chest revealed nothing abnormal.

Except for uniform bulging in hypogastrium, the abdomen is normal in contour. No pigmentation or striae, no muscular rigidity. A mass, size of a large grape fruit, is palpable in the hypogastrium. Freely movable from side to side, immovable up or down. Pressure elicits slight tenderness. The surface is firm and slightly nodular. No foetal movements, auscultation revealed no foetal heart or uterine souffle. Vaginal examination under ether showed an intact hymen, small, firm cervix, small, hard uterus not connected with firm, movable mass in pelvis.

Adnexa not palpable.

Liver, spleen and kidneys not palpable.

No X-Ray taken.

Our first diagnosis of a possible pregnancy was completely routed by the results of the vaginal examination. By a process of elimination the three most probable conditions were: First; A mesentery cyst. Second; A pedunculated ovarian cyst. Third; A Dermoid.

On opening the abdominal cavity, it was quickly discovered that none of these was correct, for the mass lay within the descending colon. An incision about six inches long was made along the border opposite the mesenteric attachment and a large enterolith, lying free in the lumen, was removed. The incision in the colon was closed by one row of catgut suture buried by silk Lembert suture. Appendix removed as routine and abdomen closed in layer suture in the usual way.

It was interesting to note that the mass lay free in the lumen of the gut, whose walls were symmetrically enlarged and not sacculated as might be expected. The symmetrical enlargement of the lumen had for these many years permitted the fecal stream to pass by on all sides and thus no obstruction had occurred. The slow growth of the enterolith had made possible the gradual adaptation of the gut wall to the unusual condition.

Various deductions may be made as to the cause of the formation of an enterolith of such proportions in a patient so young. The original cause, probably, may be found in the coarse character of the food, which formed her daily diet, and in this way she may be compared with the herbivora; but, if this were so, among the people of her native region, enteroliths should be more common. I understand that this is not the case. Her melancholia may be offered as a suggestion as to the cause, for it is well known among the aged insane, fecal impaction is common and enteroliths of fecal origin not rare. Why could not her melancholia be a secondary manifestation, due to a continued low grade of intestinal toxemia? One would expect, if this were the case, that the removal of the enterolith and daily catharsis would in time tend to improve her mental con-

dition. This did not occur, however. On the contrary she became more and more melancholic and five months later was operated upon in the City Hospital by Dr. Dawbarn, who removed a small enterolith about the size of an apple. At this time her melancholia was so profound that her attention could only be aroused with the greatest effort.

Pathological Report.

Oblong mass, about the size of a grape fruit, dark brown in color, foul odor. On the surface are seen many coarse husks of some cereal. On section no nucleus was found. The entire cut surface presented only coarse husks, seeds and stems of plants.

Measurements: 15.4 x 12 x 10 c.m.

Circumference long 43 c.m.

Circumference short 34.4 c.m.

Weight 945 grams.

Patient made an uneventful recovery and was later transferred to Bellevue Hospital because of melancholia.

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SAFETY IN THE OPERATIVE FIXATION OF INFECTED FRACTURES OF LONG BONES.*

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Accurate fixation is fully as important in the presence of infection with fractures as it is in aseptic cases. Realizing that infection nearly always prevents any foreign body from healing in and becoming encysted, I have during the past year given some attention to the selection and employment of temporary apparatus for direct operative fixation, this apparatus to remain in the tissues only until plastic exudate would make it possible to retain satisfactory position after its withdrawal.

Since perfect drainage is a sine qua non, the operative wound should be treated without closure or suture of any kind. On the contrary it had best be packed as if an acute osteomyelitis already existed. This will act either as a prophylactic against the extension of bone infection, or in the event of spreading osteomyelitis being already present it will do what ought to be done in the circumstance, that is, it will secure the drainage of the soft parts and will tend to limit the septic process in the bones.

Obviously in these infected cases an intramedullary splint of any sort is contraindicated, and the use of a Lane's plate which assures the absolute reposition of the fragment has the drawback that the removal of the plate when it shall have done its work is an operation of considerable magnitude and no little danger.

The method described by Leonard Freeman in 1904 and again before this Association in 1911 in an essay on the fixation of oblique fractures of the tibia

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and other bones by means of external clamps fastened to screws inserted into the small openings in the skin gave me a suggestive hint. As will be recollected Freeman makes use of two screws set in the bone after the reduction of the fracture and held in place by a clamp running parallel to the broken bone but outside the wound, the clamping bars being placed at the distal extremities of the screws.

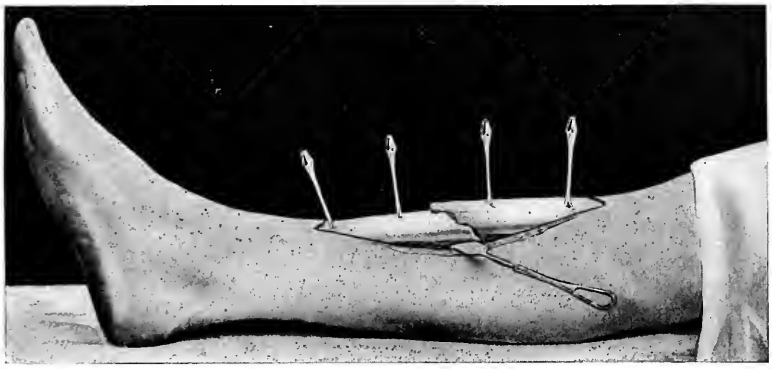
Parkhill in 1898 and Lambotte in 1907 wrote on this subject and demonstrated various forms of clamps depending on screws inserted into the bones for their fixation points. Probably the pioneer in this work was Malgaigne.

Taylor, of Port Arthur, Ontario, published a method of fixation somewhat similar to that of Freeman. Taylor put drills into the bone before placing the fragments in position and regardless of alignment. The fracture now being reduced, these drills were held in whatever position they may have assumed by one or more steel bars, placed against their distal parts and held there by a mass of plaster of Paris, thus preventing all motion in the fractured part except a very slight amount which was unavoidable on account of the moderate flexibility or spring of the drills. It has been recommended by most operators to close the wound by suture or otherwise, or else to insert the screws through mere cutaneous punctures and to remove the fixation drills at a suitable time when some union is supposed to have occurred. Doubtless in closed uninfected fractures this method would be quite proper.

During the past year I have made trial of a modification of these methods in six fractures of the long bones, four of which were closed or simple fractures and two open and more or less infected ones. The results on the whole were good as to healing and function. One case, however, in an alcoholic man with fracture of the radius, became infected and there followed some disability because of suppuration between the muscles of the forearm. I have applied the method in fractures of the tibia, the radius, the ulna, and the femur, no case being operated upon until conscientious attempts at reduction had failed. The method which I have used is one which is extremely simple in its technic and requires such ordinary tools for its successful application that I believe it deserves further trial in suitable cases.

Having exposed the fracture and a sufficient amount of bone above and below it, an ordinary gimlet of a size to fit the case is screwed into one of the fragments at right angles to the long axis of the bone. This gimlet should be fairly close to the fracture but not near enough to endanger the bone by splitting. A second gimlet is now placed an inch or so above the first one. Two other gimlets are similiarly screwed into the other fragment about the same distance from the fractured end as were the first two. The gimlet should wedge firmly into the bone so that there will be no play on gentle attempts at motion. It is not necessary to pay attention to alignment. The gimlets being in place, reduction is effected under the eye and the bone held in proper position either from without by an assistant or more directly with the aid of bone clamps. Now two pieces of steel rod about the thickness of small telegraph wire are applied in such a way along the line of gimlets and roughly parallel to the bone that the rods and gimlets shall be in contact. If the gimlets were in a perfectly straight line one rod would be sufficient, for it would touch all of them; but the line being a staggering one, two rods will be found necessary. The rods are to be placed rather far from the wound toward the heads of the gimlets. The rods and gimlets are now bandaged solidly together with a few turns of plaster-of-Paris bandage, previously sterilized by baking. In a few minutes the plaster will have set and the rods and gimlets will be one rigid mass, naturally holding the parts in the position in which they were when the plaster set. It will be found that

Fig. 1.



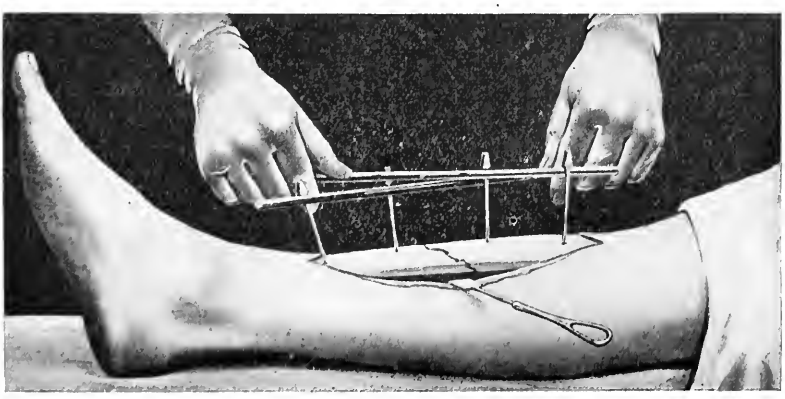
Gimlets in place; fracture not in alignment.

the bone fragments will be held in alignment by this mass of plaster of Paris, rods, and gimlets.

The wound is now disinfected and packed to the bottom with gauze, and a light rigid dressing applied. At the end of two or three weeks the gimlets will have worked loose because of slight motion and osteoporosis adjacent to the metal and may be easily and painlessly removed, the case from now on being treated as an ordinary open fracture. The gimlets which proved most satisfactory for this purpose are the ordinary square-headed variety which fit in a brace or which can be put in with a key. If at the end of two or three weeks when the gimlets are removed the wound is found to be aseptic, it may be closed with adhesive strips or sutures. Otherwise open treatment as for any infection should be pursued.

Realizing that a fixation bar or plate transmits its rigidity in inverse ratio to the distance of the plate from the bone, and that when the plate or bar is in contact with the surface of the bone rigid support will be most accurate and that for this reason the Lane plates or their modification are mechanically more

Fig. 2.



Bones in alignment; bars and gimlets in final adjustment.

Fig. 3.

Gimlets and bars being fixed with plaster-of-Paris bandage. Wound packed with gauze.

perfect than the method I have described and employed, I have devised a modification of the plating method which will combine, it is hoped, the excellent mechanical features of Mr. Lane's beautiful device without the necessity for the removal of the plates in infected cases, an operation of some magnitude.

In other words I wish to present an easily removable modification of the Lane plate and screws (Fig 8). It will be noted that at the end of the plate

Fig. 4.

Fragments held in perfect apposition by two gimlets. (Case II.)

Fig. 5.

Gimlets applied to ulna. (Case III.)

Fig. 6.

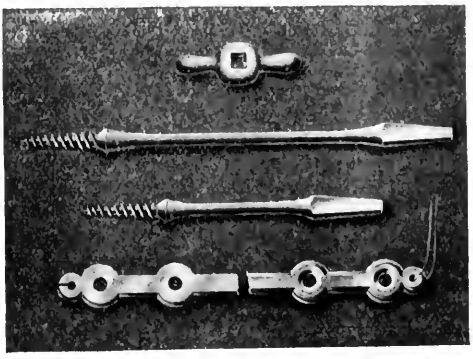
Before operation, but after attempted reposition. (Case IV.)

Fig. 7.

Slight bowing, but perfect function. (Case V.)

there is a little prolongation which is perforated to hold a stout wire. This prolongation is also filed on the bone side of the plate to form a groove so that the wire will not be compressed between plate and bone. The screws and the cone shape of their heads are similar to the Lane screws, but the head is prolonged into a shaft of from three to five inches, terminating in a square pyramidal head to fit a key which is to be used instead of the screw-driver. All the steps of the operation of plating a fractured bone will be the same as described by Mr. Lane, excepting that no screw-holder or screw-driver need be used, and that a silver plated piece of piano wire threaded into each terminal aperture in the plate will project from the wound, and that naturally these extension screw heads will also project from the wound.

Fig. 8.



Author's modification of plate and screws.

This method being in use for infected fractures, the wound itself will be packed or otherwise drained. After a suitable lapse of time, say from two to four weeks, the screws are to be removed with key or wrench and the plate is to be drawn from the wound by one or the other wire. It is expected that some force may be required for this procedure, but it is probable that the removal of the plate will be infinitely easier and speedier by this method than by an operation which means free exposure of the parts, the removal of firmly seated screws with a screw-driver, and the extraction of the plate with forceps. I repeat that it is expected that this form of plat is intended for use in infected fractures only, although it may be found on further trial that the removable apparatus may prove of value in cases not frankly infected but merely suspected at the time of operation.

In a recent conversation with Dr. Garrow, of Montreal, he gave me the history of a fracture of the femur occurring apparently through weakness of the bone due to the presence of screws in a case of a completely healed fracture which he had treated by a plating operation. The patient was a young boy and the second fracture occurred some weeks after he had been discharged from treatment. The occasion of this new fracture was a twist of the limb by a sudden turning of the body and the break occurred not at the point of original fracture but at one of the screw holes.

Now, while the plate is of tremendous value in holding firmly and accurately together the fragments of a recent or ununited fracture, it is self-evident that the mere presence of the screws in the shaft of a long bone must prove a source of weakness, because they mean the persistence of bony defects. With the screws removed these defects would fill with new bone and osseous homogeneity would be restored.

To conclude: In this paper I have tried to emphasize (1) the necessity for full drainage in all septic or suspicious fractures of long bones; (2) I have tried to demonstrate the possibility of the direct operative fixation of infected fractures while securing the necessary drainage; (3) in infected fractures direct fixation by apparatus should be temporary.

Abstracts from Case Histories.

Case I.—George C., forty-nine years old, admitted to Bellevue Hospital, August 3, 1911. Compound fracture of both bones of the leg about two inches above the ankle. Repeated attempts at reduction were vain. Operative fixation of the tibia by the gimlet method. August 24, the gimlets were removed. Afebrile recovery.

Case II.—Louis N., forty-seven years old, was admitted to Bellevue on September 1, 1911. Fracture of his left radius at the middle third. The fracture could be reduced but could not be held. Operation by gimlet method. One gimlet in each fragment. Apparatus removed on September 19, when the wound was clean, after that infection and deferred healing. Final union.

Case III.—Margaret R., fifty-three years old, admitted to Bellevue on September 15, 1911. Compound fracture of the lower part of the right radius and ulna. An X-Ray three days later showed poor position after supposed reduction Five days after injury gimlet operation on the ulna by Dr. Burdick at my request. Wound left open. Gimlets removed on October 3, and patient discharged October 13 well, but with a small healthy granulating wound.

Case IV.—Samuel W., twenty-three years old, was admitted to Bellevue Hospital September 7, 1911. Fracture of femur at junction of lower and middle third, about an inch and a half overriding. Extension failed and gimlet operation performed one week later, using one gimlet in each fragment. An X-ray

five days later showed recurrence of the over-riding through rotation of the gimlets in the bone. A secondary operation was done and two gimlets were now placed in each fragment. Aseptic healing and excellent result.

Case V.—Peter F., age ten years was admitted to Bellevue Hospital August 24, 1911. Right thigh fractured in the middle third and over-riding as shown in Fig. 7. Operation September 2, 1911, extension having failed. September 21, gimlets removed. Patient made an uninterrupted recovery.

Case VI.—Louise G., six years old, was admitted on August 11, 1911. Fracture of the fibula and the tibia in the lower third. Reduction and plaster-of-Paris dressing. X-ray showed poor position. August 24, operation by gimlets. Wound sutured between the gimlets. September 9, apparatus removed. Wound healed October 1, when patient was discharged well.

Note.—On May 20 of this year I had my first opportunity to employ the modified plate and screws which I have described in this paper. The patient was a laborer 26 years old, who had been admitted to Bellevue Hospital the day before suffering from a compound fracture of both bones of the right leg. There was a good-sized wound through which the tibia protruded. Reposition by manual effort was possible, but alignment was difficult to maintain on account of slipping of the fragments. The operation of plating was surprisingly easy, mainly because the screws could be sent home by a simple twisting motion without the slightest pressure. In using a screw-driver a certain amount of pressure is necessary in order to keep the instrument in the slot of the screw. I was astonished to note how easily and yet how solidly the long shanked screws seated themselves.

On May 22, another patient, a man 40 years old, presented himself with a compound fracture of both bones, the tibia being broken about an inch and a half above the malleolus. On May 23 I operated upon this man by the same method and was again delighted with the ease of its application. The first operation was finished in about 20 minutes, and the second in about 15 minutes. Both patients are convalescing comfortably.

I would suggest that in plating operations on subcutaneous bone the screw shanks might be made very much shorter and the idea has also occurred to me that in simple and aseptic cases in which it is desirable to follow the original method of Mr. Lane and allow the plate to heal in aseptically, the screw might be made with a square head to fit the key but without the shank.

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FRACTURE OF THE CLAVICLE—ITS DIAGNOSIS BY TRANSMISSION OF RESPIRATORY SOUNDS.*

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In a paper, read before The Society of Practical Medicine in May, 1907, entitled, "Diagnosis of Fracture of the Clavicle by Auscultation of Voice and Breath Sounds," I called attention to the fact that in normal individuals there is distinct transmission of both voice and breath sounds probably from the trachea, outward along the shaft of the clavicle to its outer extremity.

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When a small-belled stethoscope is accurately applied to the outer extremity of the clavicle, breath sounds and voice sounds of a distinctly bronchial or tracheal quality are to be heard.

In a series of over 300 normal persons thus tested, it appeared that the whispered voice was the most reliable test and that in over 95 per cent. of all the cases a more or less intensely bronchial whisper could be heard over the acromial end of the clavicle.

In over two-thirds of the cases the spoken voice as well as the whisper was of this quality, and audible. In about one-third of the cases the respiratory murmur was distinctly audible and of this bronchial character, in addition to the spoken and whispered voice.

It was also noted that the transmission of these sounds was limited very strictly to the area over the shaft and outer extremity of the clavicle, and that if the stethoscope were moved even one-half inch forward, or outward, or backward, away from the clavicle, the sound transmissions were lost or so completely modified as to offer a clear demarcation.

Conclusions: Granted that there exists normally a transmission of voice sounds from the trachea outward along the clavicle, a fracture of the bone should cause a break in sound propagation; and such is found to be the case.

In the series of fifteen cases reported at that time from the Bellevue O. P. D. the results were most striking, for, in all these cases of fracture, there was complete loss of these auscultatory signs on the injured side, in contrast to their presence on the normal clavicle. In one case of green-stick fracture, the intensity of the sounds was very much diminished although they were not entirely lost.

As bony union takes place, there is a gradual return of these signs, but this does not seem to occur until after the fourth week from the time of fracture.

In cases of prominence of the inner fragment of the clavicle, it is often very easy to place the stethoscope over this fragment and to recognize clearly the sounds which no longer are audible over the outer fragment.

Transmission of Fremitus.

Since writing the above cited paper, I have made use of a still simpler method, based on the same principles, but depended on the fact that vocal "fremitus" is likewise transmitted along the clavicle unless there be a break in the continuity of the bone.

Whereas the diagnosis with the stethoscope as above mentioned can usually be made, nevertheless there are a number of patients, especially children, who apparently cannot whisper and thus the test is rendered unsatisfactory; or there is difficulty in applying the stethoscope.

On the other hand, the recognition of vocal fremitus by the palpating finger is more easily attained and even the cries of the youngest child afford means for this test.

Method.

The examiner stands directly behind the patient, who should best be seated on a plain stool or chair. Place the thumb or index-finger of each hand on the corresponding clavicle of the patient, and without pressure.

Commencing near the sternal end of the shaft of the clavicle gradually move the examining fingers symmetrically outward along the clavicles while the patient repeats some sonorous words, e. g., "ninety-nine, ninety-nine, etc."

In the absence of a fracture the palpable fremitus will be easily detected and of equal intensity on the two sides, all the way out to the extreme end of the bone.

If there be a complete fracture, the fremitus is suddenly lost or very greatly lessened at the point of fracture and beyond. So delicately may this sign be elicited that it is often possible to follow accurately the obliquity of the line across the shaft at the point of fracture.

The normal fremitus of the whole scapular region and chest is very different in intensity and is easily distinguishable.

Value of the Method.

In many cases of fracture of the clavicle, inspection alone, or the most superficial palpation, reveals without difficulty the presence and the site of the break, and no further examination is necessary; but for the above method I claim the ease with which the signs are to be elicited, the freedom from painful palpation of the fractured bone, and its great value as an aid in the diagnosis of the cases in which the fracture is in the outer end or in the shaft without deformity, and in all doubtful cases.

Literature.

Auscultation to elicit crepitus at the site of fracture has long been in use, especially for fracture of the ribs; but in 1893, Vajana (1) of Palermo described a method for diagnosis of fractures of many of the long bones and bones of the skull by combined percussion and auscultation.

The stethoscope is placed on the subcutaneous portion of the bone and, with the plexor and pleximeter, percussion is now made at points along the bone in all directions from the stethoscope, and when a line of fracture is crossed there is loss of the sound transmission.

In 1902, J. Plesch (2) of Budapest, described the same method as original, having doubtless overlooked Vajana's communication.

A. H. Andrews (3) suggests the use of the tuning-fork instead of the plexor and pleximeter.

However, in the case of the clavicle, neither percussion nor tuning-fork is necessary, for the vocal and respiratory organs by way of the trachea, which is so close to the inner end of the clavicle, furnish all needed vibrations for our test, and, moreover, these are transmitted in a uniform direction, outward.

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CERTAIN FRACTURES OF THE UPPER EXTREMITY IN CHILDREN.*

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In this paper I shall briefly consider certain simple fractures produced by indirect violence and point out some of the practical features which are most important concerning them.

First. There is a distinct class of fractures found in children and not in adults, viz., epiphyseal separations.

While these fractures are not especially difficult to deal with as regards their immediate treatment, their later results are often very serious. Inasmuch as the growth of the bone takes place at the cartilage line, injury at this point

*Paper read and cases illustrating it presented at a Meeting of the Society of Alumni of Bellevue Hospital, January 4, 1911.

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may result in premature ossification and consequent arrest of growth. This possibility should always be mentioned to the responsible member of the family. Another feature of fracture into or through the cartilage is that the adjacent joint may be involved, an excess of bone produced about the joint and its function seriously crippled or even destroyed.

Second. We find, especially in the young, that form of incomplete break generally designated as the "green-stick" fracture. In this the bone has not been broken entirely through, but bent sufficiently to rupture the periosteum on one side with a splintering of the cortex of the bone on that side and with more or less compression or impaction on the opposite side of the bone. In this variety there are absent the usual pathognomonic signs of fracture, viz., abnormal mobility, crepitus and a false point of motion. One finds, however, angular deformity, swelling, "point-pressure" pain and loss of voluntary function.

Aside from the possible difficulty in diagnosis the practical point here is that this fracture cannot be properly reduced, as a rule, without first making it complete, because it is impossible to make the splintered ends of the fragments engage in their proper places with each other as the bone is straightened out. Unless this be done the angular deformity is bound to persist.

Third. Fractures in children are more apt to be transverse, not long splintering and oblique breaks as occur in adults. There is consequently a noticeable absence of such serious complications as compound fractures, transfixion of muscles, fascia or vessels, or extension into joints.

Fourth. Inasmuch as all physiologic and reparative functions are more active in children than adults, repair after fracture is more rapid and complete in them, with very much less likelihood of non-union than in the older.

Fifth. While there may be more or less permanent deformity due to one factor or another, after all fractures, we find the late results after this injury in children are much more perfect than in adults.

After the foregoing general consideration let us consider a few of the common fractures of the upper extremity, which derive their practical importance from their frequency and possible unsatisfactory results when not handled with due knowledge of the factors in the case.

Fractures of the Clavicle.—The only peculiarities of fractures of this bone in children are that very often the fracture is of the "green-stick" variety, or if complete is more apt to be transverse than in the adult. It usually occurs at the middle and outer thirds of the bone for well-known anatomic reasons, and is the result of an indirect force transmitted through the arm or shoulder, which in the adult usually results in a dislocation of the shoulder. While the statement has just been made that all "green-stick" fractures should be made complete as a preliminary to their reduction, when such a variety is found in the clavicle, the best treatment is to leave the fracture as it is if the deformity is slight, because if the bone is completely severed the resulting deformity is apt to be greater than without such reduction. If the fracture is complete its reduction, maintenance and results are more satisfactory than usually obtained in adults and in a shorter time, because there is less muscular development, less force to be overcome and less primary deformity. Retention of the shoulder during healing of the bone in an outward, upward and backward position is best obtained by combining a padded figure-of-eight shoulder dressing and a broad sling which passes beneath the elbow of the injured side and is tightly drawn over the sound shoulder. This dressing permits of daily inspection and correction. By no means should adhesive strapping be used for treating these

fractures in children, as, first, it is not necessary to resort to this means for satisfactory reduction, and secondly, if used it will soon excoriate the skin, become unbearable and hence useless.

Fractures of the Humerus.—Of the numerous fractures of this bone only a few will be considered, as separation at the upper epiphysis and supracondyloid fractures at the lower end. Both of these varieties possess great primary interest as regards reduction of the fracture and importance as regards the future function of the arm.

Separation of the Upper Epiphysis.—The line of the upper epiphysis runs first horizontally beneath the tuberosities until at the middle of the bone it bends downward and inward so that the surface of the epiphysis is cup-shaped, while that of the diaphysis is core-shaped to fit into it. Ossification is completed at the twenty-fifth year. A separation along the epiphyseal line is

Normal shoulder at ten years.

usually produced by indirect violence transmitted through the entire extremity, as in a fall onto the hand. The mechanism is the same as that which may in adults produce a fracture of the surgical neck or in both children and adults a dislocation of the shoulder joint.

It is due in all cases to the establishment of a condition of leverage and the formation of a false point of motion at the fulcrum. The individual falls, the extremity is thrown downward to save the body, the scapula is rotated so that its glenoid cavity is directed downward and forward to receive the impact and is fixed in this position. The hand receives the force, which tends to abduct the extremity. As the scapula is fixed this cannot take place to any great extent before the lower part of the capsule of the joint is taut and the greater tuberosity of the humerus impinges against the acromion and coracoacromial ligament. A condition of leverage is at once established and the bone gives away at its weakest point—in children at the line of the epiphysis, in adults in the same relative region; or perhaps in both the capsule of the joint ruptures first and a dislocation results. The deformity is typical and is due to muscular

action upon both fragments and gravity upon the lower one through the rest of the extremity.

The upper fragment is abducted and rotated backward by the unopposed action of the attached muscles. The inner projecting lip of the lower fragment usually lodges in the hollow of the upper fragment, forming a protuberance over the front of the shoulder somewhat simulating a forward dislocation of the joint. The diagnosis from a dislocation is settled by finding the head of the bone still in the joint cavity and its failure to rotate with the shaft of the

Normal elbow at twelve years.

Normal elbow at twelve years.

humerus. A very serious complication will be presented when this small upper fragment is dislocated outside of the joint. The reduction of the uncomplicated fracture is usually not difficult but its retention is, unless the mechanics of the problem are fully understood. The upper fragment is small and beyond the reach of the surgeon. The lower fragment is, however, under control through the arm. Apposition of the fragments is secured by remembering that the upper fragment is comparatively fixed in its position of abduction by the tension of the muscles above and the lower half of the capsule of the joint below, and

that the arm can be elevated in abduction until the shaft is brought into line with the upper fragment, complete apposition being obtained when the bicipital groove in each fragment is made continuous. The extremity is then fixed in this position of abduction and slight external rotation by a plaster spica which envelops the chest and upper extremity as far as the wrist.

Union will take place in three or four weeks and the functional result is usually almost perfect.

The only objection advanced against this position of abduction of the arm is its awkward appearance and possible discomfort to the patient. While the positon is certainly awkward to look at and for the patient to endure there is no other discomfort attending it. They get about, sleep and lead a perfectly happy existence.

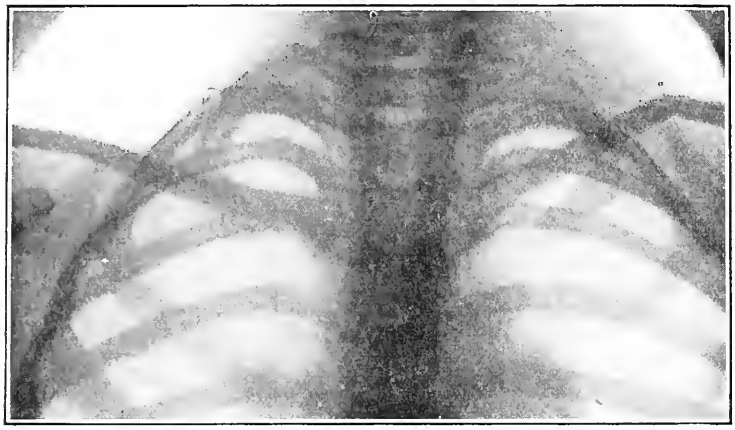
At times reduction of the fracture can only be secured in some complicated cases through an open incision, and excision of the epiphysis in such

Normal wrist and hand at ten years.

cases may be necessary, but as this paper is limited to the simple cases these complicated instances will be omitted.

Supracondyloid Fractures.—These fractures are within the lower inch-and-a-half of the humerus. While there are a great many variations in the line of the fracture we shall confine ourselves to a consideration of the so-called "extension," "flexion" and "adduction" forms of fracture.

The more common form is the **"extension" fracture,** in which the line of fracture is oblique from above downward and forward. The injury is produced by a fall onto the hand or forearm, the elbow is fixed in a position of flexion, the body acts as the weight to drive the humerus downward and forward, the lower end of which is fixed by the position of the forearm; the two forces meet at the elbow and a break occurs at the lower end of the humerus through the disassociation of these forces. The line of this fracture is from above downward and forward, the "extension" form of supracondyloid fracture.



Greenstick fracture of clavicle (six years).

If adduction takes part in the mechanism, as is often the case, the fracture is usually at a lower level and an obliquity added from the outer condyloid ridge downward and inward.

If in falling the arm be more fully flexed and the force directed backward, the line of fracture may run from in front and above to downward and backward, producing the "flexion" variety of fracture. In the "adduction" form the line of fracture is directed from the outside and above downward and inward, the lower fragment consisting of a greater or less amount of the external condyle and the bone immediately above it. In addition to these fractures there may be a great number of variations added which we cannot consider at this time.

The recognition of these varieties is assisted by remembering and interrogating the position of the bony points about the elbow, by knowing that the

Epiphyseal separation of head of humerus, before reduction (eight years).

Same after application of plaster spica Abduction was carried a little too far.

"extension" fracture presents some of the characteristics of a dislocation of both bones of the forearm backward, also that the traction forward on the forearm which reduces the deformity in the above increases it if the fracture is of the "flexion" variety.

An exact diagnosis in fractures about the elbow is possibly more imperative than elsewhere on account of the disabling results of imperfect reduction and deformed union being here more serious as regards the comfort and usefulness of the patient than is the case with fractures anywhere else in the body.

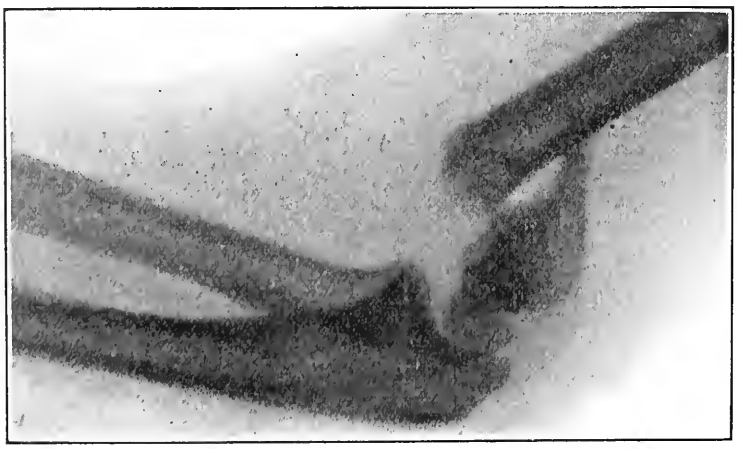
Therefore it is necessary that all means for making a diagnosis be utilized,

Same case after abduction was lessened. Union was firm in this position at the end of three weeks, with apparently perfect anatomical and functional result.

Fracture just below the surgical neck of the humerus (eight years).

viz., interrogation of the bony point with and without an anesthetic and X-ray photographs taken in both vertical and horizontal planes. Fractures which extend into the joint not only add to the difficulties of the diagnosis by the excessive amount of swelling that follows but also to the difficulty of obtaining the best results on account of the callus which forms in and about the joint, limiting its range of motion. It is never safe, then, to predict the final result with too much assurance, as Nature's reparative forces are so little under our control. There is one undesirable result which, however, I think we should consider within the range of our art to prevent, and that is recovery with loss of the "carrying angle"—a deformity both disfiguring in looks and with defective function of the limb.

The usual method of treating these fractures is by the application of moulds or splints embracing the shoulder and forearm with the elbow at the side. If



Transverse supracondyloid fracture of the humerus (eight years).
The so-called "extension" fracture.

The same anteroposteriorly. The excessive deformity does not show in this view, hence the necessity of taking X-ray photographs in two planes at right angles to each other. There is total loss of the "carrying angle."

The same elbow after extension had been made in the abducted position and plaster spica applied. Shortening entirely overcome.

The same case viewed anteroposteriorly, taken at the same time as the preceding photograph.

The same case after the elastic traction on the forearm had been in use for three days. Result anatomically and functionally perfect.

there is little tendency for the lower fragment to be displaced forward or backward, and the deformity is solely an overriding of the fragments, this method will give good results, provided continuous extension is made downward during the day by a weight suspended on the forearm close to the elbow while the wrist is fixed by a narrow sling that keeps the forearm flexed at right angles to the arm. Under no circumstances should the sling extend backward to embrace the elbow; by so doing there will be produced the very deformity you are seeking to prevent, viz., loss of the carrying angle. This

Fracture of the external condyle of the humerus.
The "abduction fracture." (Age nine years.)

result is caused by the patient resting the weight of the arm in the sling. Naturally this brings the greater weight upon the ulnar side of the fragment, and by the steady pressure it is crowded upward more on this side than on the other. Union resulting in this position leaves the trochlea on a level with the capitellum and the normal angle of abduction is lost and the gun-stock deformity substituted.

While the extension by weight acts satisfactorily during the day with the patient up and around, it cannot be used if the patient is in bed by day or night.

A good substitute is found through the means of a weight and pulley, or by the use of a short axillary crutch. The long arm of the crutch extends some six inches below the elbow, and to it is fastened an elastic band connected to a strap over the forearm close to the elbow. Whatever form of traction is used it must not be excessive; slight, steady, continuous force being more efficient than severe intermittent traction.

In some cases, however, where the direction of the fracture is from above and behind downward and forward, or where there is comminution of the lower fragment, this method does not give that close apposition of fragments and consequently the best results so earnestly desired. Therefore I would suggest the method of treating these fractures with the arm in the abducted position.

Extension and apposition of the fragments is secured, with the patient under a general anesthetic, by making extension on the flexed forearm at right angles to the chest. A plaster spica is then applied, embracing the chest and fixing the arm in this position of right-angled abduction to the chest—and the

Fracture of the olecranon process. (Fifteen years.) Separation of the fragments due to flexion of the forearm.

forearm at right-angled flexion to the arm. This position allows of extension and counter extension being maintained against the forearm and side of the chest without any danger to the axillary contents. Before the plaster splint is applied to the forearm, two moleskin adhesive strips, one to two inches wide, are applied to the dorsal and ventral surfaces of the forearm. These adhesive strips reach to the palm of the hand. While applying the splint to the forearm, two narrow strips of thin board (as from a cigar box cover) an inch wide are incorporated into the dorsal surface of the splint. These strips reach to the end of the fingers. When the plaster has set the ends of the adhesive strips are attached to the ends of the wooden strips by strong rubber bands. This traction is not excessive, causes no discomfort, but it acts continuously to overcome any posterior displacement of the lower fragment. By this method the carrying angle is preserved and owing to the effective fixation of the parts the formation of excessive callus is prevented and the attendant dangers of subsequent joint interference reduced to a minimum.

I am not in favor of treating fractures about the elbow in the extended position, though I recognize the fact that in this position you have control of the forearm and through it of the lower fragment and can fix the angle of abduction. However, one cannot prevent the backward tilting of the fragment in this position, and recovery will be followed by deformity and loss of function, especially in flexion.

I have not utilized the method of treating these fractures by acute flexion of the elbow and fixation of the hands on the shoulder of the sound side, as advocated by Jones and several other writers, because my results with other

"Greenstick" fracture of the radius (eight years).

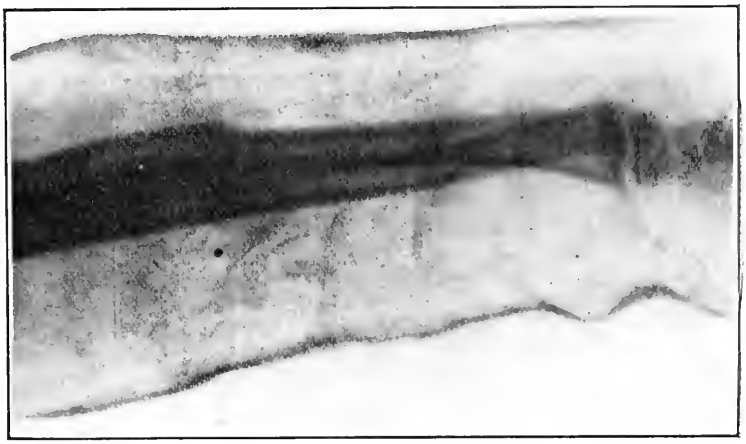
methods have been satisfactory. I should expect, though, that this method would produce good results in the ordinary varieties of the above fractures of the humerus.

It would seem superfluous for me to reiterate the warnings contained in all the text-books against applying roller bandages directly to the extremity immediately after fractures or applying a tight roller bandage to the limb at any time on account of the danger of producing superficial sloughs or even strangulation of the limb sufficient to cause Volkman's ischemic paralysis, or even total gangrene of the extremity. But judging from our experience, from the cases

reported in the medical journals from time to time, and from the frequency with which such cases are aired in the courts, there seems to be a real need to repeat these warnings and cautions.

Fractures of the Forearm.—The most frequent variety consists of the green-stick fracture of one or both bones. The usual cause is a fall onto the hand, and due apparently to a very trivial force. The lesion is usually at the middle and lower thirds of the bones. The objective symptoms are the deformity, point-pressure pain, swelling and loss of function. The interest centers about this fact, that unless the fracture is converted into a complete one it cannot be fully reduced, hence the necessity of completing the break.

As to the method of splinting simple fractures of the forearm I prefer to fashion my own splints out of any light board, as a cigar-box cover. These



Same case from the side. The deformity was subsequently corrected with a perfect result.

board splints, as Stimson emphasizes, must be wider than the diameter of the limb and well padded. I do not extend the splints beyond the wrist in any of these fractures, not even a Colles, because such extension is not necessary for maintaining the position of the fragments once they have been properly placed in line; second, immobilization of the joints of the wrist and fingers for two or three weeks may produce such stiffness in these joints as to require a longer time for the recovery of their function than is required for the healing of the fracture. Stopping the splints at the wrist allows the patient to begin exercising all the distal joints from the very first, so that when the splints are removed there will be normal motion in these parts.

I accept Stimson's verdict that interosseous splints are useless, because when put on tight enough to accomplish their object (separation of the fragments of the radius and ulna in fractures of both bones), they cannot be borne, and if not put on as tight as this they accomplish nothing and hence are useless.

Fractures of the olecranon are produced in the majority of cases, not by a fall onto the elbow, but by a fall onto the hand with the forearm flexed. The olecranon being fixed by the contraction of the triceps forced flexion of the forearm by the weight of the body snaps the olecranon at its weakest point, its middle, and then the patient's elbow strikes the ground.

The recognition of this fracture is easy. The amount of separation of the

fragments depends more upon the flexion of the forearm than the traction of the triceps. If the forearm is fully extended no difficullty will be experienced in bringing the tip in contact with the shaft. Adhesive strapping over the tip of the upper fragment with the forearm extended is usually sufficient to secure close apposition and a firm union. If fascia or muscular elements intervene between the fragments, as shown by the impossibility of bringing them closely together and securing sharp bony crepitus, it is proper to remove this obstacle and to suture the fragments through a vertical incision with kangaroo tendon

Impaction fracture in the lower end of the radius. Either this form or a separation of the lower epiphysis constitutes the "Colles fracture in a child (nine years).

or chromic gut sutures passing through the fibrous structures. It is not necessary, therefore, nor wise, to drill the bones, and a suture of unabsorbable material is not required.

Separation of the lower radial epiphysis producing the symptoms of a Colles fracture requires no special consideration because of its occurrence in a child. It requires the same complete reduction of the deformity that the corresponding injury in the adult does, and as a rule gives more satisfactory results. However, the possibility of premature ossification and consequent arrest of growth in the radius must be entertained in the prognosis.

I have been asked to state my position in regard to the use of metal

splints or other means, as nails, drills, screws, wire, etc., for the direct fixation of fractured bones.

To my mind their use can only be justified by the exceptional case, almost exclusively found in the adult, such as compound fractures with great deformity, fractures of the neck of the femur with great displacement of the fragments, or in cases of non-union of the large, long bones where correct apposition cannot be maintained without such appliances.

In any case, especially in children, the mere correction of a deformity does not justify one to resort to such severe means as the open method, with its real danger of infection, even under the most rigid asepsis, when sufficiently accurate apposition can be obtained under the use of an anesthetic and by appropriate splinting with traction applied patiently and intelligently during the first two weeks of any fracture in a child to secure a good functional result. Reparative processes in children are so rapid and bony irregularities so constantly and satisfactorily smoothed off and obliterated that the simpler and less dangerous methods give results nearly perfect anatomatically and quite so functionally.

In passing, I might state that I have had to remove several of these bone plates, which had been placed in position by others more enthusiastic in the use of these metal splints than myself. Further, some of my colleagues, who formerly were partial to the use of these splints in simple fractures have after their more mature experience discontinued their use.

In all cases of fracture if in doubt give an anesthetic to assist in making the diagnosis, and if possible also seek the aid of the X-ray photographs. This will conduce to accuracy of diagnosis, more intelligent treatment of the fracture and better final results.

Before concluding this paper I wish to extend my thanks to Dr. William H. Stewart, Radiographist of Harlem, Fordham and Gouverneur Hospitals, to whom I am indebted for the radiographs of my own cases and others from his collection to complete the series.

THE SURGICAL TREATMENT OF IRREDUCIBLE DISLOCATIONS OF THE SHOULDER AND ELBOW JOINTS.*

Lucius W. Hotchkiss, M.D.

Articular surgery, as applied to the relief of old irreducible dislocations of the larger joints, only became a possibility after the introduction of the antiseptic wound treatment of Lister, and its development corresponds closely with that of the modern aseptic surgical technic. From the first successful case of tenotomy of the tendon of the pectoralis major, performed by Weinholt in 1819, in the reduction of an old dislocation of the shoulder, to the operations of the present day, the history of arthrotomy affords a fascinating record of progress and achievement, in a class of cases long held to be beyond the range of surgery. The first successful case in America of arthrotomy for dislocation of the elbow, was reported by Warren of Boston in 1869, and later, numerous cases here and abroad were reported by Langenbeck, von Lesser, von Wahl, Ollier, Hamilton, and many others. In 1892 Stimson published his studies of the anatomy of old elbow luxations and demonstrated clearly the feasibility of reducing them after an arthrotomy with division of restricting bands and generally without joint resection or a preliminary division of the olecranon.

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Reprinted from New York State Journal of Medicine, September, 1912.

Stimson's work made clear many points in the pathology of old elbow dislocations which had not been hitherto understood; and in addition to clearing up the diagnosis, placed the treatment by open arthrotomy with reduction upon a rational basis.

Although we speak of these irreducible luxations as "old" or "ancient," it must be remembered that a considerable number of these cases are irreducible primarily, and so may be neither old nor ancient.

There are, therefore, two great groups of cases to be considered:

First, those which for various causes are irreducible from their inception, and second, those which though primarily reducible, have become irreducible through the lapse of time and the growth of adhesions.

In the first group, the conditions which may prevent primary reduction are numerous and variable. Thus the head of a displaced radius may be thrust through an overlying muscle and so firmly gripped as to be irreplaceable without excision of the head of the bone, as happened in one of my own cases. Or the head of the humerus may be restrained by an interposed tendon or muscle, from being returned to its proper place without division of the restraining bands in question.

Or there may be a complicating fracture of the head or neck or process of the bone which by its displacement renders reduction difficult or impossible without resection.

In the second, and by far the more numerous group, the growth of adhesions, the contraction of the aperture in the torn capsule, the contraction and adhesions and atrophy of the surrounding muscles and soft parts secondary to the trauma and to the pressure upon important nerve trunks, the adhesions of important blood vessels to the torn capsule of the injured joint, all tend to make reduction by manipulation not only difficult or impossible but extremely dangerous.

The possibility of causing a fracture of the humerus in elderly people by forcible attempts at reduction of an old shoulder luxation constitutes a real danger, which is not always heeded; and the possibility of rupture of an important nerve or vessel from the heel in the axilla or from over forcible manipulations in these old cases, is ever present, and cases of this sort do not all belong by any means to prehistoric times.

It will be evident, then, inasmuch as the exact conditions existing in any given case of old irreducible luxation of an important joint can only be ascertained by a direct inspection through an open incision, viz., an arthotomy, that such an operation is a rational procedure.

The only other questions to be answered, then, are, is there such an operation? is it effectual? and to both of these, I think, an affirmative answer may be returned. A large number of cases has been reported and the results are generally satisfactory. The mortality of the operation, according to Dollinger, who has collected a large number of records of his own and from other sources, is about 2 per cent., i. e., about the same as the mortality from dislocations of joints of the upper extremity which have been unoperated. This low mortality rate seems surprising in view of the severity of the operative procedure in some cases, and speaks volumes for the reliability of modern surgical methods.

In this brief paper, which is purely practical and based largely upon the personal results obtained in the operative treatment of 15 cases of old dislocation of the shoulder and elbow joint, the writer has confined his attention to these two articulations because the conditions in question are perhaps most often

observed therein. Of course, similar operative treatment is equally applicable to the other large joints.

My personal records of arthrotomy for old irreducible dislocations of the shoulder and elbow, include 15 cases, of which 8 were of the shoulder and 7 of the elbow.

In these 15 operated cases, there was no mortality.

The ages of the patients varied between 7 and 60, and in all save one, in which the record is incomplete, the functional results were good. One must always expect, perhaps, some stiffness following an arthrotomy at the shoulder with division of extensive adhesions and replacement of the bone to its normal position. This is generally more than compensated for, however, by the movable scapula; while the restoration of the rotundity of the shoulder and the absolute relief of pain, in cases where this was a marked feature, give much satisfaction and relief to the patient.

As a rule the diagnosis is easy in the old shoulder dislocations and may often be made by inspection alone, so characteristic is the deformity in some cases. An X-ray plate, however, clears up many unsuspected conditions, and is most useful both as a guide and as a record. In the old backward dislocations of the elbow, on the other hand, the differential diagnosis between it and fracture of the condyle, is by no means always clear and here the X-ray is especially valuable and necessary.

As to the operation itself, arthrotomy consists essentially in an incision made into the affected joint for the purpose of determining what had best be done with that joint. The further procedures consist in the division of adhesions, the careful conservation of certain tendons and the nerve and vascular trunks, and in the reduction of the joint surfaces to their normal positions, or, if this be not practical, in the performing of an excision of bone as the best means of securing the best results. It is evident, then, that as much depends upon the judgment of the surgeon in these cases as upon his operative skill, to solve the problems which may suddenly present themselves. As in all my cases of unreduced dislocation of the shoulder the head of the humerus was displaced anteriorly, I have had only experience with the anterior incisions, viz., that of Andrews dividing the fibers of the pectoralis major directly across and resuturing the same at the end of the operation, and with the older incision, which runs along the upper humerus between the borders of the deltoid and the pectoralis major. With the axillary incision, which is said to give an admirable exposure of the field, I have had no experience. With the incision of Andrews I have been well satisfied, as it gives a wide and direct exposure of the field of operation and gives a better approach than the longitudinal incision in these particular cases.

Although an arthrotomy for the reduction of an old dislocation of the shoulder appears a somewhat formidable procedure, it is not, in my experience, as difficult as an arthrotomy at the elbow for the reposition of both bones backward. In this latter form of luxation, of which there were four in my series of cases, the approach to the joint is best made by the method of Stimson or through the antero-lateral incision described by Kocher. Although some of the forward and partial dislocations may be dealt with through other incisions, the method of Stimson, if carefully followed, best enables one to open the elbow joint, clean out the olecranon fossa, chisel off the new growth of bone caused by the tearing up of the periosteum above the internal condyle of the humerus and to deal with the various constricting bands and adhesions with the least danger to the integrity of important structures about the joint. For a full description of the incisions and the methods of dealing with the conditions found,

those who are interested are referred to the original article of L. A. Stimson on this subject. My first case of arthrotomy in an old unrecognized and unreduced dislocation of the elbow in a man of over 50 was operated upon by the method in Bellevue Hospital in 1892, shortly after the publication of Stimson's paper, which had been the inspiration of my first work. In the case of old unreduced elbows the older surgeons generally preferred excision of the joint. This operation indeed is much easier and much more quickly done, but a reductio ad integrum, after a careful arthrotomy gives results which, I think, are superior as to strength and utility.

To this very brief description of the possibilities and scope of a carefully planned arthrotomy in old unreduced dislocations of the joints of the upper extremity, I append the abbreviated histories of my individual cases.

Histories. I. Elbow.

No. 1.—Backward dislocation of both bones of the forearm. Unreduced elbow had been put up in full extension and patient was helpless as regards use of joint. Duration, several weeks. Operation, Bellevue Hospital, 1892. Arthrotomy, Stimson's incision, cleaning out of olecranon fossa, etc.; reduction, closure with drainage. Result, good flexion, but considerable lateral mobility which did not interfere much with usefulness.

No. 2.—Child, female, age 9. Backward dislocation of both bones of the forearm. Arthrotomy, reduction, Hood Wright Hospital, July, 1895. Dislocation five weeks old. Posterior incision, division of restricting bands, reduction. Good functions and motion. Reported N. Y. Surgical Soc. Trans. Annals of Surgery, Vol. 22, p. 798.

No. 3.—Child, female. Outward dislocation at elbow. Arthrotomy, reduction, Hood Wright Hospital, February, 1901. Good functional result.

No. 4.—Male, adult. Exploratory arthrotomy, irreducible dislocation of elbow, Hood Wright Hospital, February, 1902. Result not known. History lost.

No. 5.—Irreducible forward dislocation head of the radius. Male, 24, Roosevelt Hospital, May 18, 1905. Excision of head of radius which was thrust through the overlying muscle; orbicular ligament torn. Good function.

No. 6.—Old backward dislocation both bones of forearm at elbow. Adult, male. Arthrotomy, reduction. Difficult. Had been treated as fracture and put up in full extension. Reduction perfect; good function; some stiffness yielding to massage; functional result excellent. Operation at Roosevelt Hospital, February 4, 1909.

No. 7.—Boy, age 7. Roosevelt Hospital, October 6, 1909. Anterior dislocation elbow, both bones forward; complication, fracture external epicondyle and capitellum fractured. Replaced without suture; dislocation reduced; short opertion. Good function.

II. Shoulder Cases.

No. 1.—Old subcoracoid, irreducible by manipulation. Arthrotomy, reduction. Good function. Adult, male, Hood Wright Hospital, October, 1898.

No. 2.—July 28, 1903, Roosevelt Hospital. Old subcoracoid, ten weeks. Difficult arthrotomy. Division long head of biceps and external rotators. Posterior edge head of scapula splintered. Male, 28. Good function. Seen one year later. Abduction limited, function good.

No. 3.—Old subcoracoid, very painful from pressure on nerves; one year from date of injury. Arthrotomy reduction. Relief of pain; good function. Adult, female. Roosevelt Hospital, September 2, 1903. Reported to Surgical Society.

No. 4.—Old subcoracoid. Bellevue Hospital, May 27, 1905. Male, 50. Arthrotomy. Reduced after very extensive division of adhesions. Excision of humeral head below tuberosity, which showed evidence of old fracture. Good function; useful arm; resumed work.

No. 5.—Bellevue Hospital, January 31, 1908. Old subcoracoid. Male, 60. Andrews' incision. Arthrotomy, reduction difficult and after wide division. Resection head of humerus. Good function.

No. 6.—Old injury, dislocation complicated with comminuted fracture of head of humerus; dense adhesions; excision of the head of the humerus. Andrews' incision. Good result. Female. Bellevue Hospital, January 31, 1908.

No. 7.—Old subcoracoid. Bellevue Hospital, April 9, 1910. Adult, male. Arthrotomy, Andrews' incision. Reduction; subscapular muscle divided and resutured with capsule. Good function.

No. 8.—Old subcoracoid. Bellevue Hospital, June 6, 1911. Arthrotomy, reduction. Good result.

OPERATIVE TREATMENT OF FRACTURES*

JOHN W. WALKER, M.D.

Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; Surgeon to Bellevue Hospital.

In June, 1909, before this Association at Philadelphia I read a paper on the "Operative Treatment of Fractures," hoping that an increasing number of members would become more interested in developing the operative treatment for selected cases of fractures; two members discussed it. Two weeks later at Atlantic City, before the American Medical Association, Mr. Lane presented a paper upon the same subject. There were few who discussed it. The general sentiment prevailed that the average results after fractures were satisfactory and that operations were unnecessary. In 1911, at Denver, President Harte in his address called special attention to this subject. He stated his belief that careful observation of the end results after fracture justified the conclusion that these results were not all satisfactory as was generally believed, and moreover that our profession was not fully utilizing all the advantages in our power to secure the best results in fractures; therefore, he advised the operative treatment for selected cases, especially in the femur.

In 1909, there were only a few papers on this subject, while in 1912, the literature was full of references to operations. Numerous operations have now been performed by many of our own members and as their experience has increased they have gradually operated earlier and more often because they have obtained better results. Many surgeons are feeling at present that numbers of the "bad set" fractures which become useful only after twelve to eighteen months might be just as good after four to six months if more efficiently treated. These facts show clearly the great increase in the amount of attention paid to securing better results after fractures. During the last two years, as our profession has been more carefully investigating the end results after fracture, the surgeons have become convinced that the results must be improved. The public, since the advent of X-rays, have become better educated and are demanding shorter and more efficient treatment, a briefer period of disability with better functional results.

Feeling that the reporting of illustrative cases will do more to encourage

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other members to undertake the operative method in selected cases. I desire to present the following: As the fractures of the femur are the ones which most frequently demand and finally come to operation, and also as they present the greatest difficulties at operation, the favorable results which can be obtained in these cases should carry corresponding weight in determining which is the best method of treatment in definite selected cases.

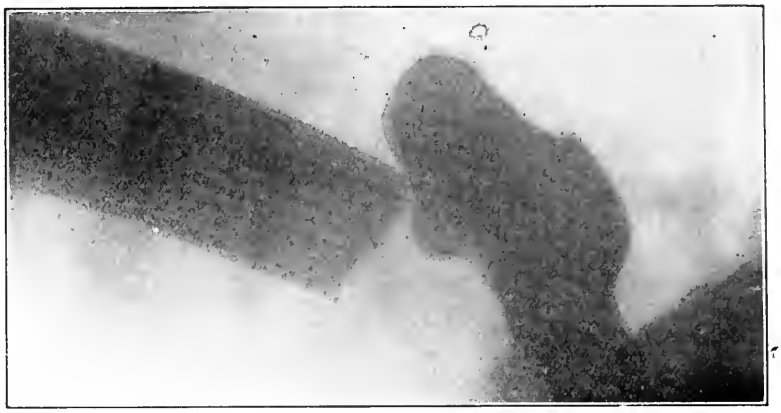
In this series of 21 cases of fracture of the femur, operation was performed only after the best efforts of conservative treatment had failed. In every case before operation general anaesthesia had been employed to assist the efforts in reduction, also suitable extension had been applied. Nevertheless, in every case there persisted over 2.5 cm. shortening. Axial rotation was present in all cases, together with angulation.

In operating, I try to scrupulously carry out every minute detail of Lane's technic, for there is no province of surgery in which results depend more upon the mechanical skill and cleanliness of the operator. The day before the operation the part is thoroughly washed and a dry sterile dressing applied. On the day of operation the patient is etherized and then placed upon a platform raised above the operation table, so arranged that the Lemon extension apparatus can be used. The sterile dressing is removed and the entire part painted with 7 per cent. tincture of iodine. The usual operation sheets are placed to carefully exclude all surfaces except the immediate operative field, which is again painted with iodine. The incision, which is always long, from 8 to 10 inches, is made on the outer aspect of the thigh, being carried through the vastus externus and crureus. To both edges of the skin incision strips of gauze are at once clamped. As the incision is carried through the muscles to the bones, all free bleeding points are firmly clamped but not tied, the clamps acting by their weight as retractors. The clamps are left on the vessels for some time while the other dissecting is continued. Bleeding generally ceases, but if it continues, ligation is done with the finest catgut. The tourniquet is never used, as it is necessary to see at once when any sizable vessel is cut. The tissues are treated gently so as to avoid all tearing, lacerating, and consequent bleeding. Great care is taken to clearly expose the fragments; granulation tissue is cut away with scissors; callus is removed with the rongeur. The ends and edges of the fragments are curetted clearly so as to permit them to fit into each other as closely as possible. The periosteum is protected as much as possible and is not stripped from the bones. In manipulating the ends of the bones to bring them into position I prefer the Lambotte clamps to those of Lane, because they are easier to handle and are self-retaining. After the bones are in position, the Lohman clamp is often most satisfactory in holding the plate securely to the fragments.

Thus far, I have used the regular Lane plate. I have never seen one broken, although cases have been reported. In the future I hope to use the vanadium plates introduced by Dr. William O'Neill Sherman, of the Carnegie Steel Company, if they can be made stronger, lighter, and smaller. In some earlier cases, I tried both wire and kangaroo tendon, but found them absolutely unsatisfactory in fractures of the femur. Lane uses the old-fashioned drill, because its simplicity permits it to be thoroughly and easily sterilized. The handles of the usual twist drills cannot be so easily cleaned, so I have used the Stille drill. Every part is detached after an operation and thoroughly cleaned and sterilized. The drill most used is No. 31 calibre standard twist. This makes a hole slightly smaller than the size of the barrel of the screw. The screws are the ordinary wood screws modified so that the thread is cut up to the head. I prefer them to the finer threaded machine screw because the threads are coarser and seem

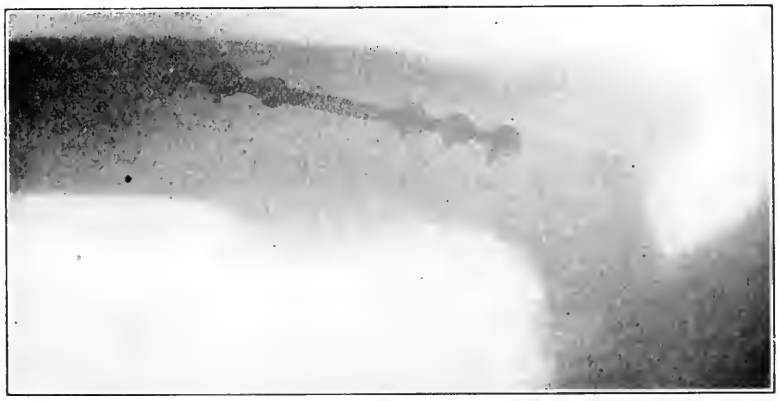
to hold better in the varying densities of bone. The screws do not go through the entire diameter of the shaft of the femur, but only through the cortex to which the plate is applied. During the operation the blood is removed by dry gauze sponges, but the tissues are not rubbed. No irrigation is employed. Under no circumstances whatever do the fingers ever enter the wound. The separated layers of muscles and fascia generally come together because they are under tension, so no sutures are required. Further, this method freely permits the escape of the oozing which is always present. The skin edges are brought together by interrupted black silk sutures which are put in place and tied with forceps. No drain is used. A very thick absorbent dry dressing is put on, and

Fig. 1.



Femur, upper third. (Case XI A.)

Fig. 2.



Femur, upper third. X-ray taken two years after operation. Normal function regained within one year after operation. (Case XI B.)

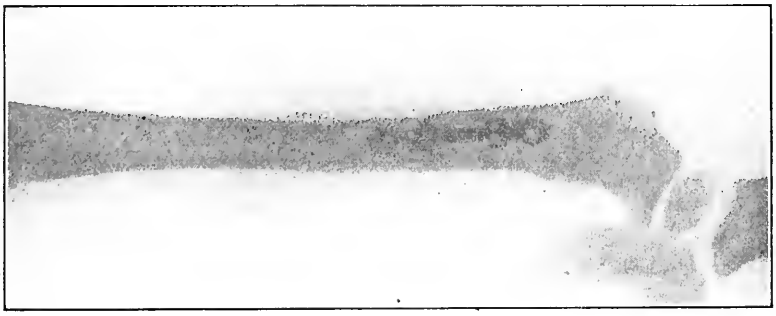
over this is applied a plaster case strengthened with wood or steel splints, extending from the toes to and embracing the pelvis. The patient remains upon the table until the case becomes solid.

After the strong plate has been most satisfactorily applied by snug screws to the shaft of the femur it would seem as if no motion were possible. If, how-

Fig. 3.

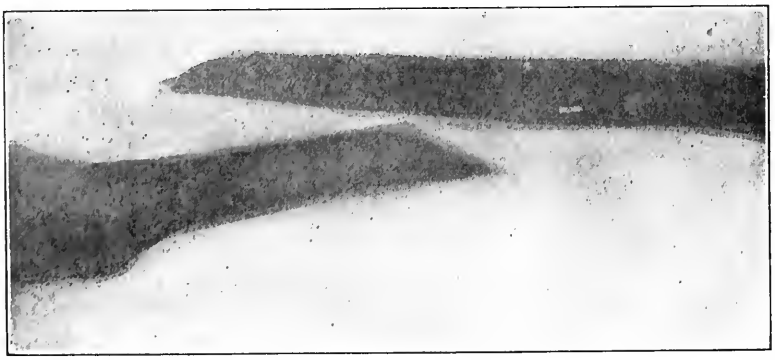
Femur, upper third. (Case X A.)

Fig. 4.



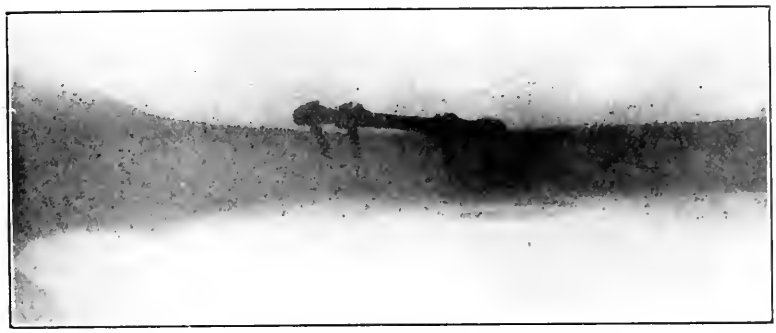
Femur, upper third. X-ray taken two years after operation. Normal function regained within one year after operation. (Case X B.)

Fig. 5.



Femur, middle third. (Case VIII A.)

Fig. 6.



Femur, middle third. One month after operation, slight superficial suppuration plate removed. Normal function regained within six months after operation. (Case VIII B.)

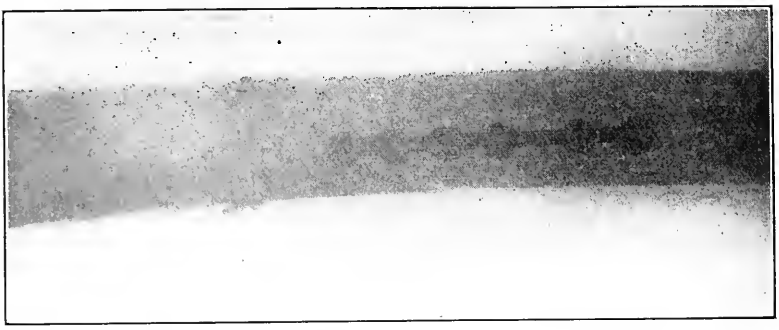
Fig. 7.

Femur, middle third. X-ray taken three years after operation. (Case VIII C.)

Fig. 8.

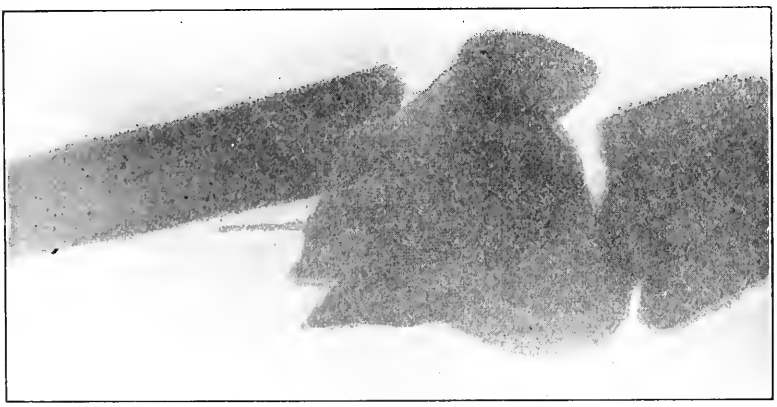
Femur, middle third. (Case XIII A.)

Fig. 9.



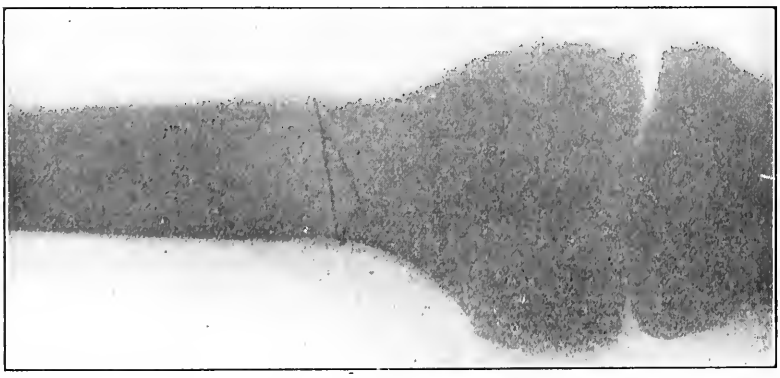
Femur, middle third. X-ray taken one year after operation. Normal function regained within four months after operation. (Case XIII B.)

Fig. 10.



Femur, lower third. (Case VI A.)

Fig. 11.



Femur, lower third. X-ray taken one year after operation. Function much improved within one year after operation. (Case VI B)

Fig. 12.

Femur, lower third. (Case XX A.)

ever, moderate strain be applied to the leg, some motion at the fracture can be appreciated. If this be continued the screws will become loosened and the fragments disarranged. For this reason no strain must be permitted. The plate must be considered only of value merely to approximate the fragments and not at all sufficient to hold them. For this purpose the whole reliance must be placed upon the solid external plaster case, most accurately and carefully applied. If this does not succeed in absolutely immobilizing the fragments, the operation may fail.

Before undertaking such operations it is especially necessary to provide a distinct armamentarium, for success here depends so absolutely upon employing just the right instruments. It has been found very unsatisfactory to depend for traction upon the efforts of our assistants, and frequently their combined strength has failed to reduce the fragments. I am sure that those surgeons

Fig. 13.

Femur, lower third. X-ray made six weeks after operation; primary union. Patient on crutches in hospital. Result excellent. (Case XX B.)

who have had an extended experience all thoroughly appreciate the necessity of having powerful traction which can at any moment be applied or discontinued; it must be sufficiently flexible in its working to permit at first a small amount of traction, which later can be easily increased when more traction is required. During the last six months I have been using Dr. Lemon's extension apparatus and have found this of the greatest possible assistance in those late cases where, after several months had elapsed, there existed marked shortening, with rigidity of the muscles and the adjacent tissues. The leg is held steadily during the operation, and when it is completed, the plaster case can be easily applied without releasing the extension. It can also be maintained until the case becomes solid. This has given me great comfort in relieving my anxiety lest the fragments become displaced when the patient is first moved from the table after the operation.

There has been no mortality. In only one case was the plate removed and that was in one of my earlier cases, when I was somewhat apprehensive, but when I cut down to the plate the screws were solid and it would have been unnecessary. "If these statistics leave something to be desired, it must be remembered that these were cases picked as being too difficult for other forms of treatment and that our city hospital patients are frequently poorly nourished and of the worst habits." Operations performed under the above indicated methods have been followed by excellent results. If this is possible in the cases of old, long-standing difficult fractures of the femur, how much more easily and more quickly can it be done in recent cases and with how much greater safety and surety of securing an earlier and better functional result. Fractures of the femur comprise about 7 to 10 per cent. of all hospital fractures and are most difficult to treat. Surgeons are interested in establishing the best method of treatment for the different special fractures. It now appears that sufficient evidence has been shown to definitely recommend operations for fractures of the femur, in such cases as reduction is inadequate. Adequate reduction requires that the ends remain in apposition without obvious angulation or axial rotation, and that the shortening be not greater than one-half inch. In every case skilled efforts must be made to secure reduction at once after the accident. If these efforts are unsuccessful, they must be renewed under general anaesthesia. If adequate reduction is secured and can be maintained by traction, extension apparatus is applied. Further efforts to secure reduction by extension should not be continued after seven days, as it has been frequently demonstrated, that where over-riding could not be pulled down in that time, no benefit could be gained by longer traction. Repeated violent attempts have caused more traumatism to the tissues than the operation itself. Some observers have stated that distinct changes occurred early in the ends of the fragments which caused delayed union as the bone channels became blocked up with lime salts.

Case number	Year of operation	Age	Seat of fracture				Reduction attempted under anesthetic	Extension applied	Cm. shortening	Angulation persisted	Rotation present	Days elapsed after accident before operation
			Neck	Upper third	Middle third	Lower third						
1	1900	40	..	..	+	..	+	+	3.5	+	+	420
2	1906	28	+	..	..	..	+	+	3	+	+	795
3	1907	12	..	..	+	..	+	+	4	+	+	39
4	1908	36	..	+	..	..	+	+	4	+	+	30
5	1908	48	..	+	..	..	+	+	4	+	+	60
6	1908	52	..	..	..	+	+	+	3½	+	+	98
7	1909	40	..	..	+	..	+	+	4½	+	+	47
8	1909	10	..	+	..	..	+	+	2½	+	+	13
9	1909	11	..	..	+	..	+	+	3	+	+	14
10	1909	16 mos.	..	+	..	..	+	+	2	+	+	485
11	1910	42	..	+	..	..	+	+	4	+	+	6
12	1910	60	..	+	..	..	+	+	2½	+	+	14
13	1911	43	..	..	+	..	+	+	4	+	+	10
14	1911	50	..	+	..	..	+	+	3	+	+	5
15	1911	36	..	+	..	..	+	+	5	+	+	7
16	1911	50	..	..	..	+	+	+	3½	+	+	31
17	1912	34	..	+	..	..	+	+	3	+	+	35
18	1912	12	..	..	+	..	+	+	2½	+	+	37
19	1912	50	+	..	..	..	+	+	2	+	+	325
20	1912	34	..	..	..	+	+	+	3	+	+	193
21	1912	57	..	..	+	..	+	+	3	+	+	78

There has been no mortality or serious complications; in only one case has the plate been removed, No. 8. Improvement has resulted in every case.

Conclusions.

1. In fractures, the rapidity of union is proportional to the accuracy of reduction and the retention of fragments; delayed union is very largely due to faulty adjustment.

2. Plating the fragments does not increase the nutrition, but it brings the fragments into early intimate contact.

3. "As our experience grows we will be able to select after a study of the X-ray plates those cases in which operation is indicated." This will save the patient loss of time in trying extension, and will also render the operation easier, safer, and surer.

The accompanying illustrations represent the typical class of cases which demand operation in order to obtain the most satisfactory results.

THE SURGICAL TREATMENT OF MENINGITIS, ITS SCOPE AND ACCOMPLISHMENT.*

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Chapter I.—Introduction.

The term "meningitis" was introduced by an army surgeon, named Herpin (1), in 1803. It indicates a septic process involving the membranes of the brain caused by various organisms. The mortality in meningitis varies from twenty per cent. to one hundred per cent., depending upon the kind of infecting organism. Its lethal effects are due to three separate and yet inseparably intermingled factors, the poisonous effects of the toxins manufactured by the bacteria themselves, the toxins generated in the tissues and cerebro-spinal fluid as a result of bacterial action upon them, and, to the mechanical effects of the pressure produced within the skull by the products of inflammation. Septic conditions are usually surgical opportunities and demand surgical treatment.

The treatment of suppuration within the meninges of the brain is not on a par with the treatment of sepsis elsewhere in the body. In most all other forms of septic infection, special or general, local or diffuse, surgery has secured a great diminution of mortality and a remarkable preservation of function. But, concerning meningitis, with the exception of the serous and diplococcic forms, its mortality is just as high as it was in 1870, before the dawn of cranio-spinal surgery, which since then in other lesions of the brain and cord had achieved such notable results.

I propose to review the records of the past forty years, to ascertain, if possible, the reasons for the present deplorable situation in the treatment of this disease and to show that there is hope for the future.

After correlating the experiences of the past, the opinions of the present and the latest results in practical and laboratory research, with a firm belief in the reason and justice of our contention, I desire to submit for critical examination and actual test a rational, safe and efficient surgical procedure for the treatment of meningitis.

Historical Review.

As a preliminary observation in regard to the operative treatment of intracranial suppuration the following quotation from a paper by Bross (2) is given; this was published in 1873:

"The treatment of compression of the brain from effusion of pus is purely surgical, and all authors are unanimous in the opinion that, unless the matter

be evacuated, the patient will die, although they are equally agreed that this measure holds out but little chance of life."

A little further he says: "No case is recorded of recovery from "effusion of pus into the arachnoid cavity," that one feels that he left out * * * on the other hand, if a free incision be made into the dura mater so as to admit of its ready escape, one-half of the patients, as I have just shown, fight their way through. The necessity of operation is, therefore, not to be questioned."

While Gross referred to intra-cranial suppuration due primarily to traumatic causes, his statement is so explicit in reference to "effusion of pus into the arachnoid cavity," that one feels that he fully comprehended the necessity of surgical measures in all such cases of whatever origin, and their utter hopelessness when otherwise treated.

We go a long ways before we come to anyone else suggesting surgical intervention for "effusion of pus within the arachnoid cavity."

Ventricular puncture and drainage is the next deliberate procedure offered for the relief of the effusion in meningitis.

According to von Beck (3), this conception was first expressed theoretically by Wernicke, in 1881.

Further that von Bergman was the first to use it in 1888, in a case of tubercular meningitis. He was closely followed by a number of operators, as Keen, Robson, Franks, Wyss, Hahn, Kocher and McCosh, in such cranial lesions as meningitis, brain tumors, and epilepsy.

Von Beck reports a case of "Acute Internal Hydrocephalus" following a chronic otitis media, in a boy of 14, in which the lateral ventricles were punctured at three different times, with the withdrawal of 26, 40, and 40 ccm. of cerebro-spinal fluid. The child recovered.

He also described a case of "A Traumatic Cerebral Abscess" in the frontal lobe that was incised and drained. This was followed by diffuse, purulent meningitis with acute internal hydrocephalus. Fifteen days after the first operation the lateral ventricle was punctured, 40 ccm. of turbid cerebro-spinal fluid drawn off. The boy (7 years old) was moribund at the time of the last operation, but recovered after an illness of 126 days.

Quincke (4) describes a case of a child, 12 years old, with hydrocephalus, upon whom he performed ventricular puncture on June 25, 1888, because all other forms of treatment had been unavailing. The lateral ventricles were tapped at different points five times thereafter, but the child finally died from meningitis.

W. Mayo Robson (5) was among the first to conceive the idea of ventricular drainage in meningitis, and, while antedated in its execution by both von Bergman and Quincke, is entitled with them to receive credit for this operative suggestion. He writes:—"In 1888, while sitting powerless by the bed of a child dying of coma, due to meningitis, a train of thoughts passed through my mind, which culminated in the ideas set forth in this paper. If we have a serous, purulent or tuberculous peritonitis, we do not hesitate to open the abdomen and drain it; and if we have a pleura full of fluid, we feel it our duty to perform paracentesis, or if the fluid be purulent, to incise and drain the chest. Then why do we allow the brain to suffer compression, even unto death, when we can, as the following case will prove, tap the ventricles and remove the pressure?" Here follows the report of a case, in which he operated on February 7, 1889, by puncturing the lateral ventricles through a trephine opening over the left motor area. Six ounces of clear fluid were removed. Recovery followed, without superficial suppuration, in a month.

The plan next suggested and executed for relief in meningitis, attacked the

disease at an entirely different point, namely, by draining the spinal subarachnoid space in the lumbar region.

To W. Essex Wynter (6) must be given the credit for the conception and first operation of spinal drainage in the treatment of meningitis. His first case was operated upon February, 1889.

Of course, to Corning (7) belongs the honor of the first lumbar puncture, but this was for the injection of drugs, not for drainage of inflammatory exudation.

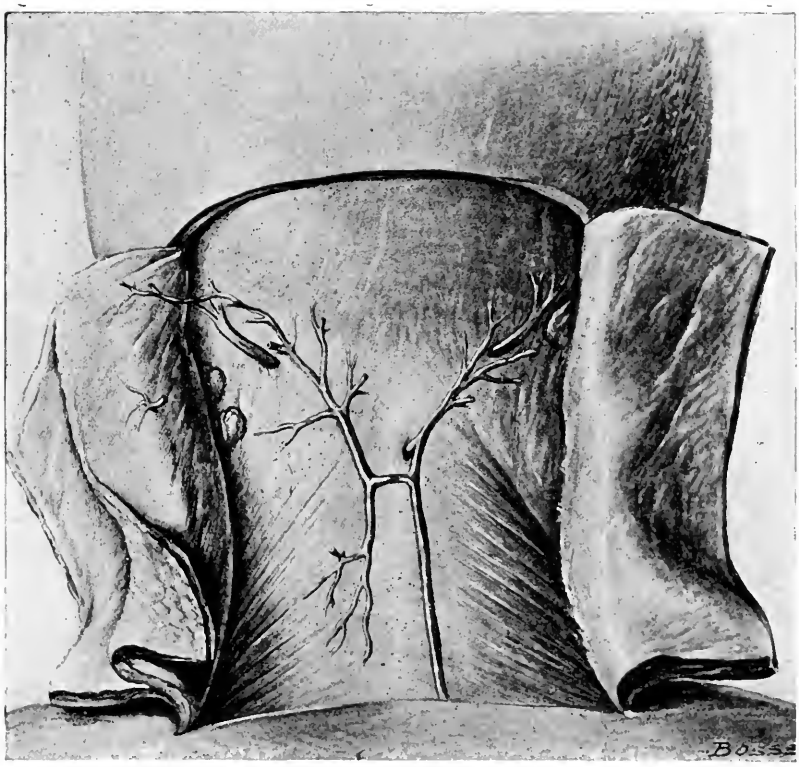
In his paper, Wynter says: "In many cases of fatal tubercular meningitis, coma supervenes with great rapidity and with all the signs of cerebral pressure, whilst after death little can be found except an excess of cerebro-spinal fluid, often at a considerable positive pressure, to account for the symptoms and the fatal issue. The possibility of recovery in this class of cases, where there is commonly little development of tubercle, together with the feasibility of draining away the fluid continuously by the theca vertebralis, suggested this mode of procedure."

His first case was a boy of 3 years of age in the comatose stage of tubercular meningitis. The operation did not require an anesthetic. The child was held in a sitting position. "A tiny incision was made in the skin beside the spine of the second lumbar vertebra and a Southey's tube and trocar inserted till the point impinged against the lamina; the point was then directed slightly downward and was pushed through the ligamentum and theca with an inclination toward the middle line. Clear fluid at once welled up into the tube on withdrawing the trocar, a fine India rubber tube was arranged for continuous drainage and the child put back to bed." Although all the symptoms were immediately improved, the fluid ceased to flow after twenty-four hours and the child died.

Wynter's second case, operated upon February 21, 1890, was a lumbar laminectomy. Briefly, it is as follows: Girl, aged 11. Wynter cut down upon the second lumbar vertebra, removed the spine and right lamina and punctured the theca with a knife. Clear fluid escaped with force. A drainage tube was placed in the canal. Improvement in all symptoms followed immediately, but with the stoppage of the flow of fluid three days later, they became worse and the child di d on the next day.

It was not until December 12, 1890, that Quincke (4) performed lumbar puncture according to a technic devised by himself and accepted and universally practiced ever since. It is due to his careful experiments and explicit directions for performing this operation that his claims for pioneer work in this field rests, because he was clearly antedated by Wynter by a year and ten months, in the actual performance of the operation.

Please note that Wynter's first case was a deliberate lumbar puncture with provision for continuous drainage, whereas his second case was a lumbar laminectomy with tube drainage. Quincke's first case had very marked hydrocephalus, with acute cerebro-spinal meningitis. A boy, 18 months old. Quincke did his first lumbar puncture December 12, 1890, between the third and fourth lumbar vertebrae and drew off a cubic centimeter of clear fluid. The puncture was repeated on December 14, with removal of 10 ccm. fluid in 20 or 30 minutes. Again on December 17, puncture below the fourth vertebra was done and the pressure of the cerebro-spinal fluid noted at 13 to 15 cm. of water, or 10 to 11 mm. Hg. When the child cried the pressure rose to 20 cm. of water but quickly dropped back to 15 cm. again. The flow was about 8 drops per minute. The child was convalescent by December 23, and fully recovered by January 10. Quincke considered this a lepto-meningitis infantum or simple acute hydro-



Figures 1, 2 3. Dissections by the writer.
Drawn by Mr. Bosse, reduced.
Veins injected to make them more prominent.
Fig. 1.—Dissection of the post-cervical region superficial to the trapezius muscle. A small anastomotic vein is shown crossing the line of the incision.

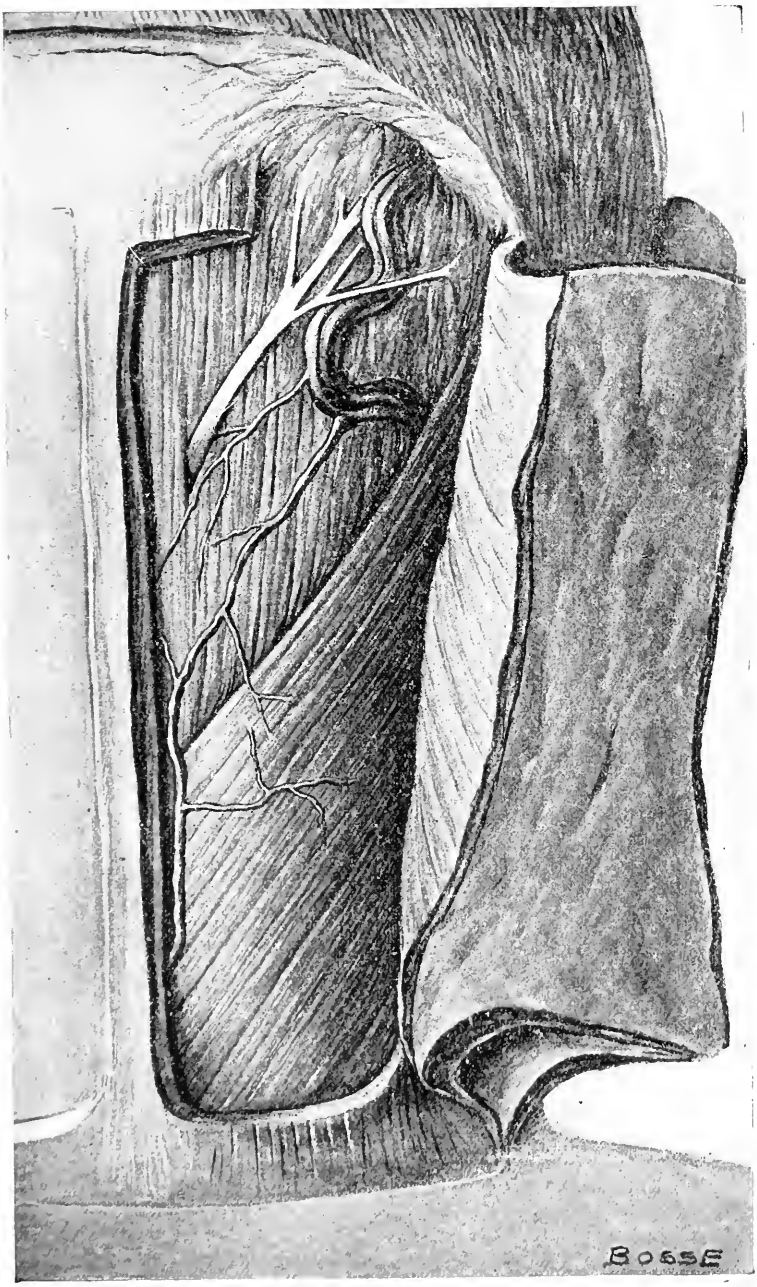


Fig. 2.—Structures superficial to the complexus muscle.

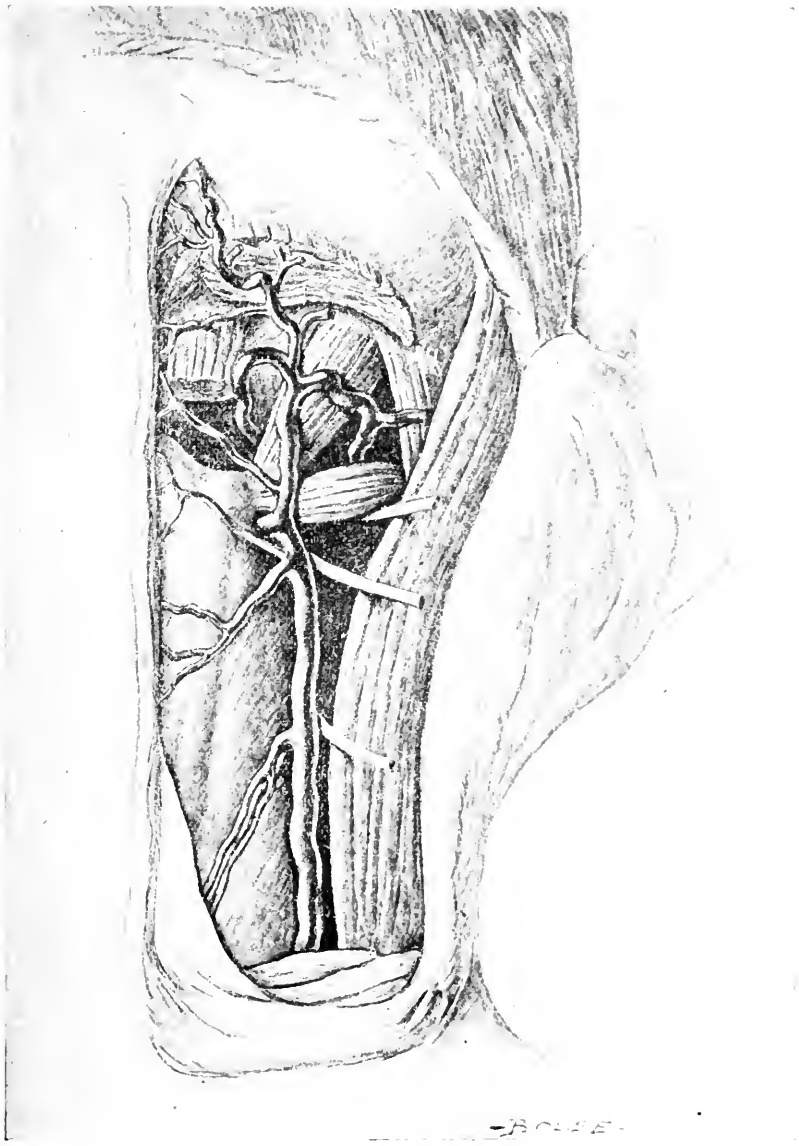


Fig. 3.—The deeper structures. The small anastomotic veins which cross the line of incision.

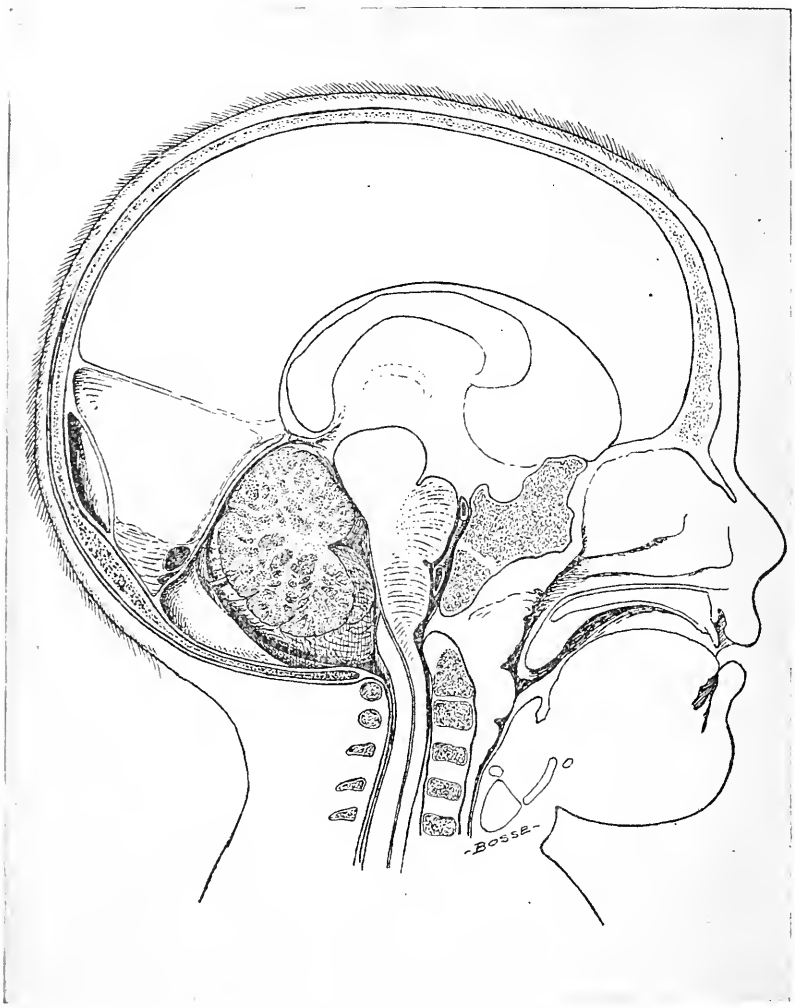
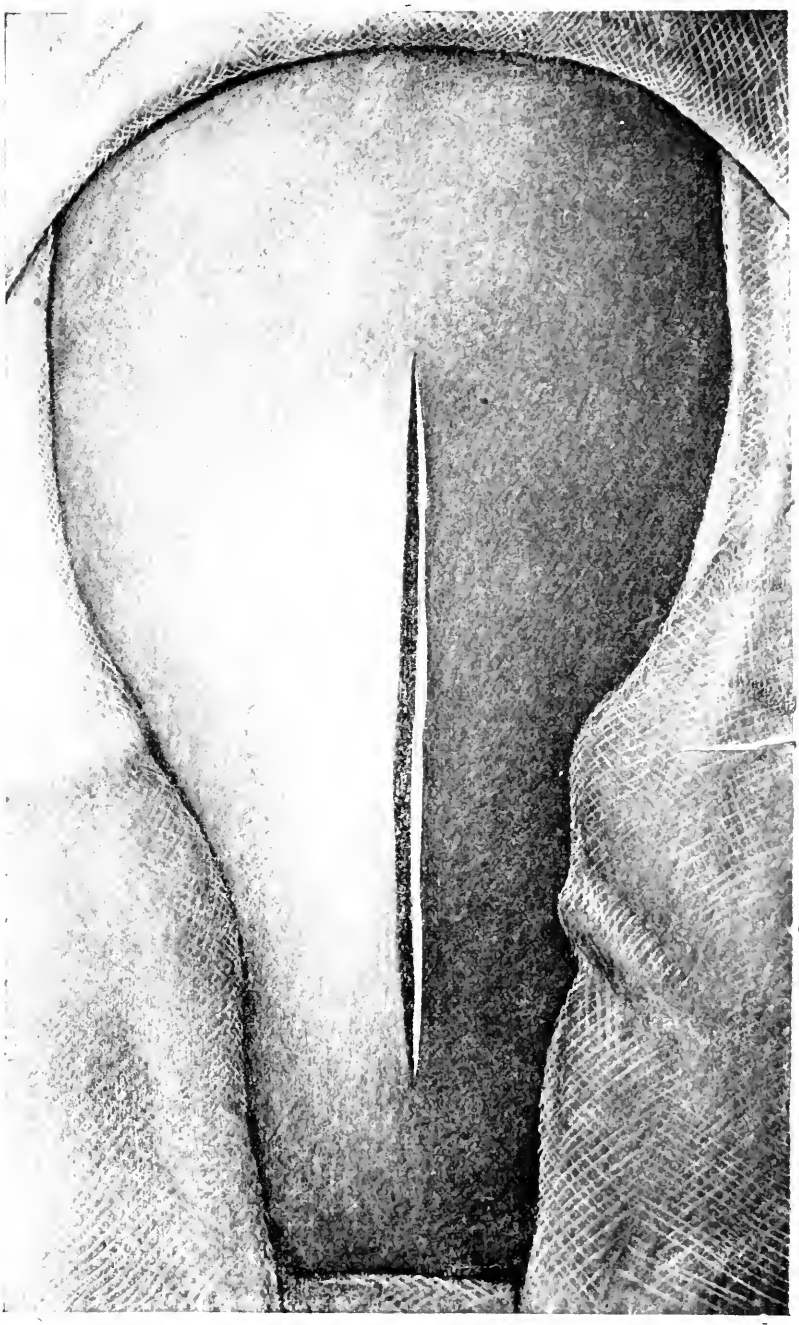


Fig. 4.—Saggital section of the head and neck of a child. The subject had been hardened in formaldehyde and sectioned by the writer; (slightly reduced).

Note.—Vertical position of brain stem. Its relation to the foramen magnum. The ease with which "corking" this foramen could occur.

The large cisterna magna, its relations to the other subarachnoid spaces and especially to the occipital bone near the foramen magnum. Also the very narrow space between the foramen magnum and the arch of the atlas.

The distance of the skin from the occipito-vertebral angle is greatly diminished by flexion of the head and spine.



Figures 5 to 12. Illustrate the steps in the operation of "Draining the Cisterna Magna."

Preparations by the writer upon an adult male subject. Figures 5 to 8 are slightly reduced. The remaining, on a scale twice that size.

Fig. 5.—The incision.

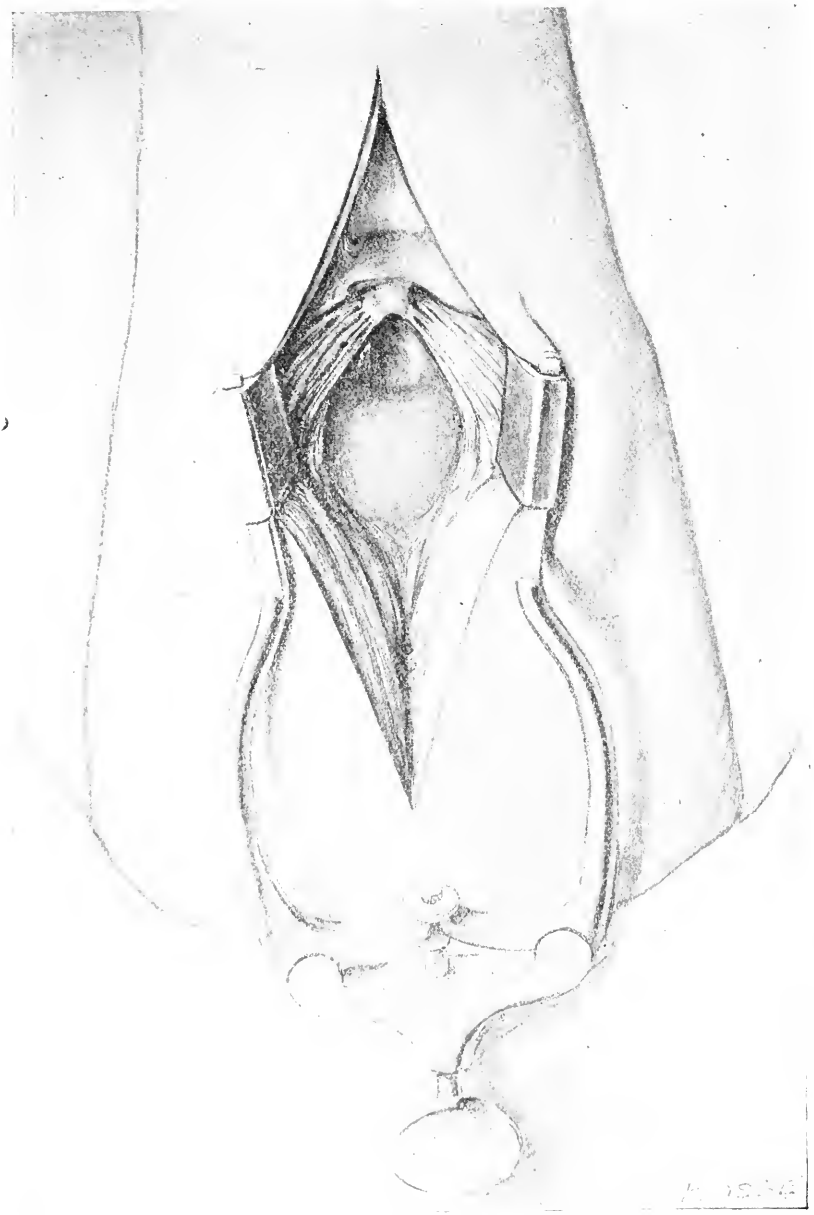


Fig. 6.—The incision has been carried down to the occipital bone and posterior arch of the atlas. The pericranium with the muscles has been reflected from the occipital bone. The field is fully exposed by the aid of the self-retaining retractor.

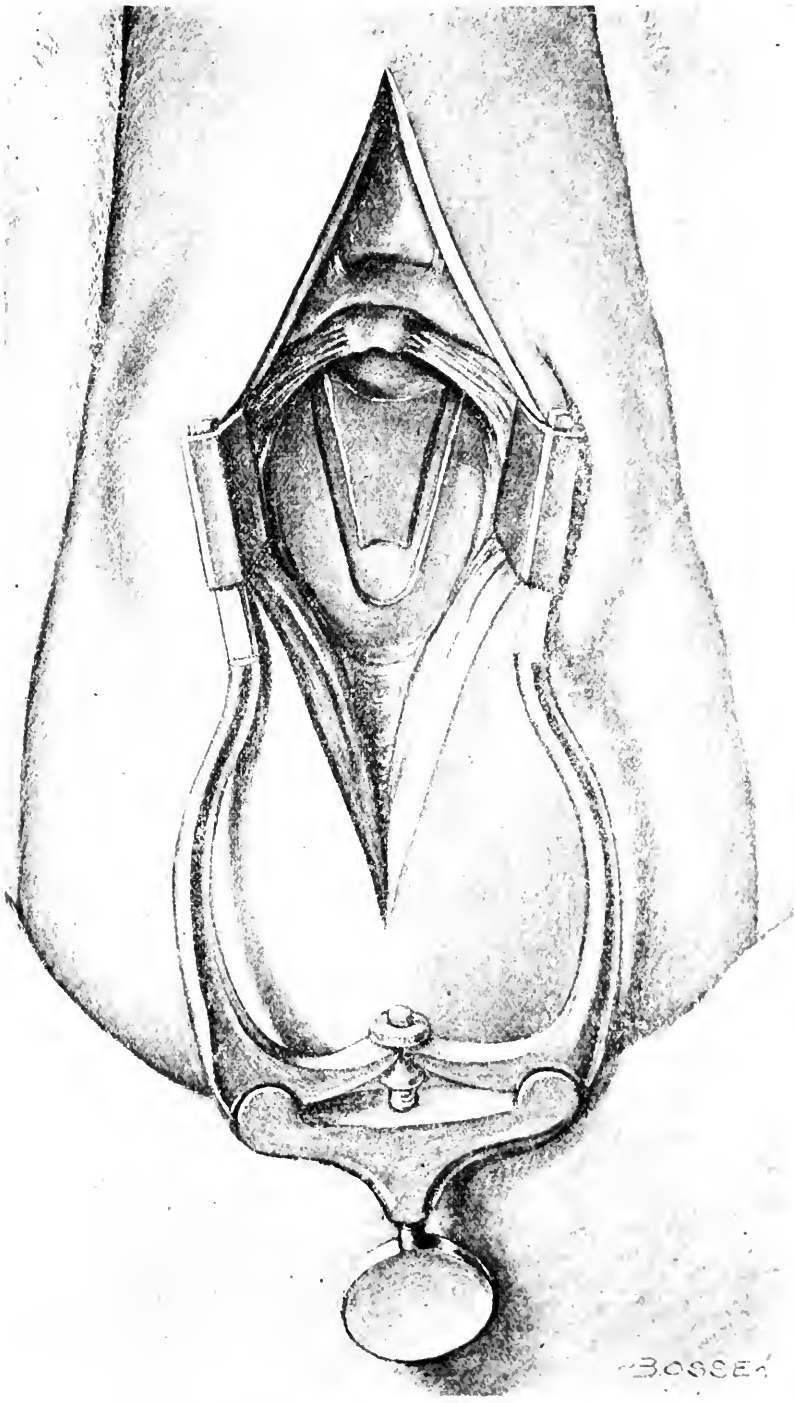


Fig. 7.—The trephine opening has been made and grooves have been cut into the foramen magnum.

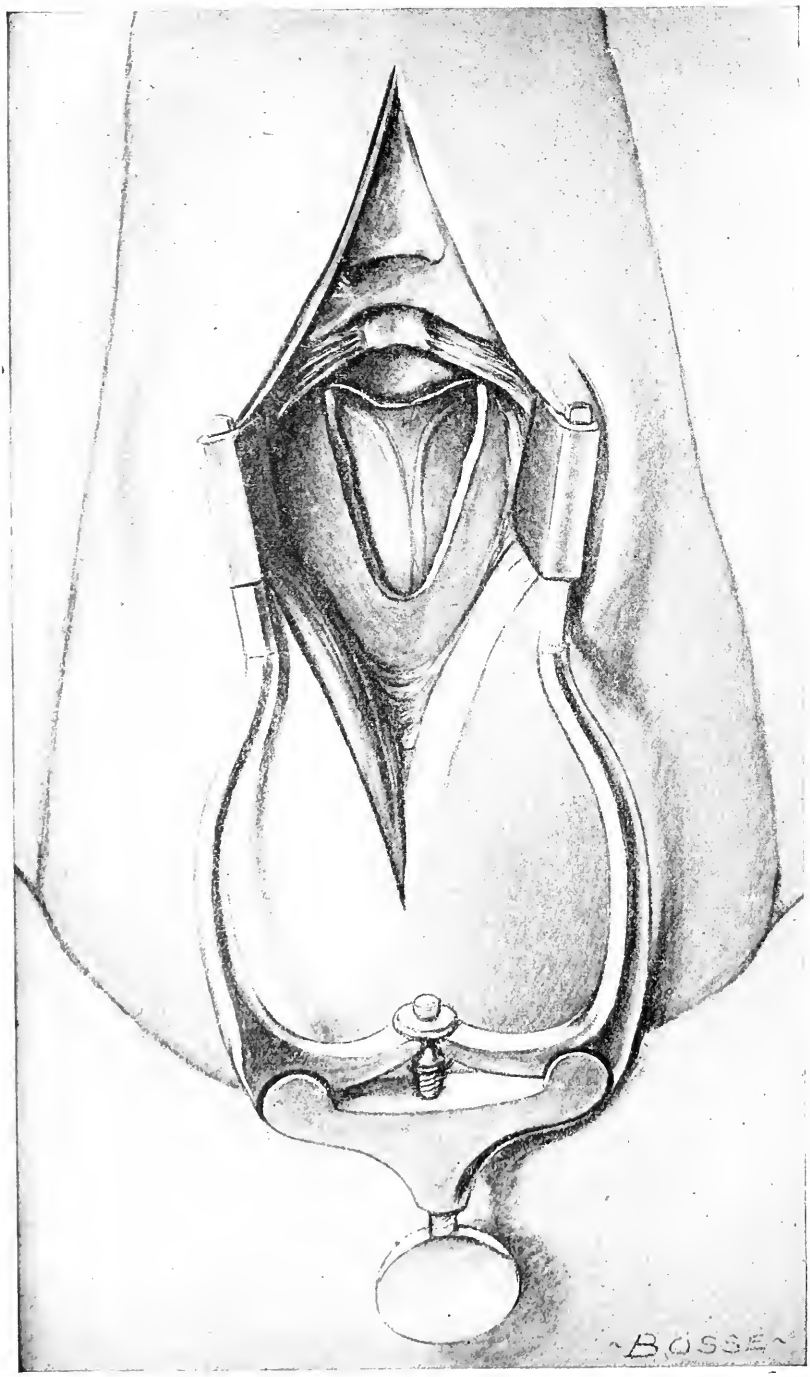


Fig. 8.—The wedge of bone has been removed. In this cadaver the occipital sinus was double, as shown in the illustration, and both clearly visible.

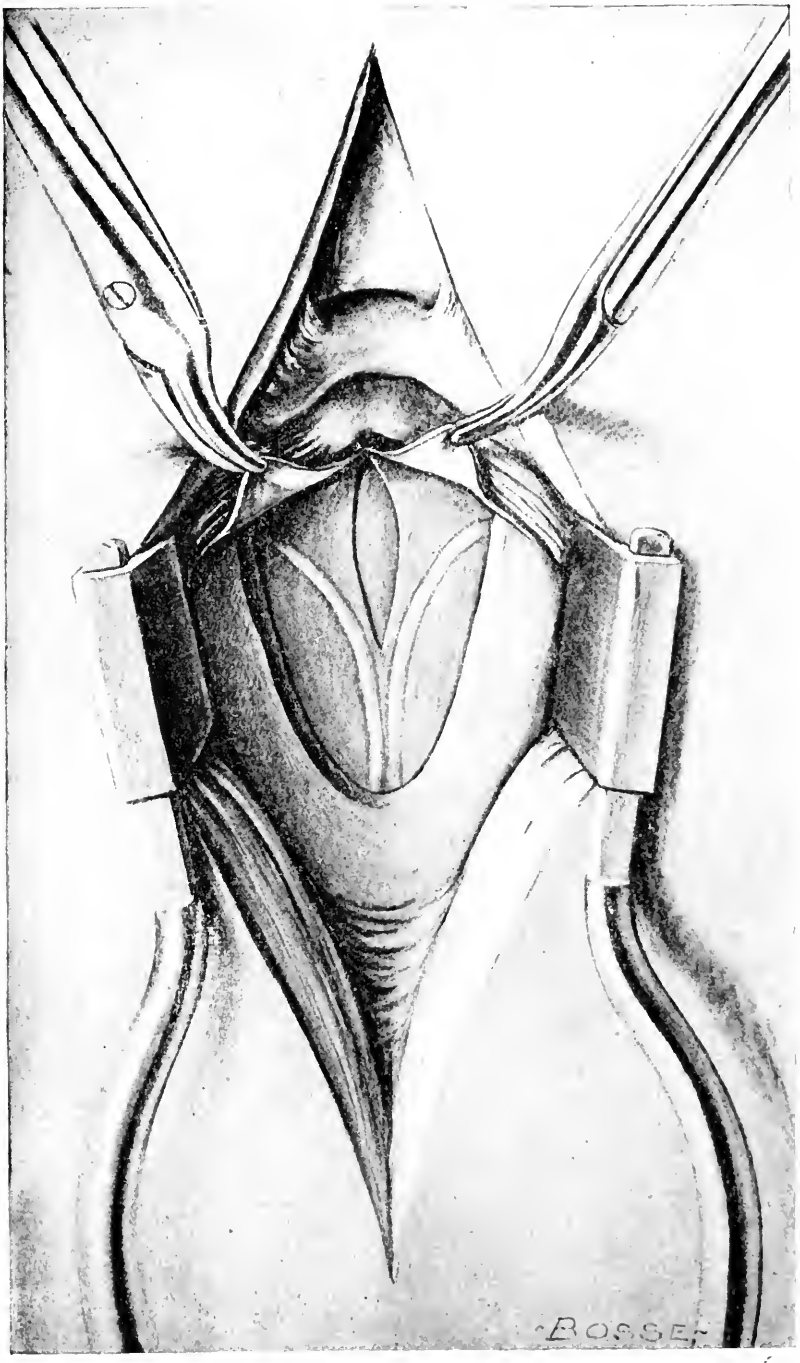


Fig. 9.—This figure and the two following have been drawn twice the natural size to render the structures more distinct. The dura has been opened between the double occipital sinus and the arachnoid is presenting.

Fig. 10.—The arachnoid had been divided and the cisterna magna opened. Note the space between the lobes of the cerebellum and the medulla.

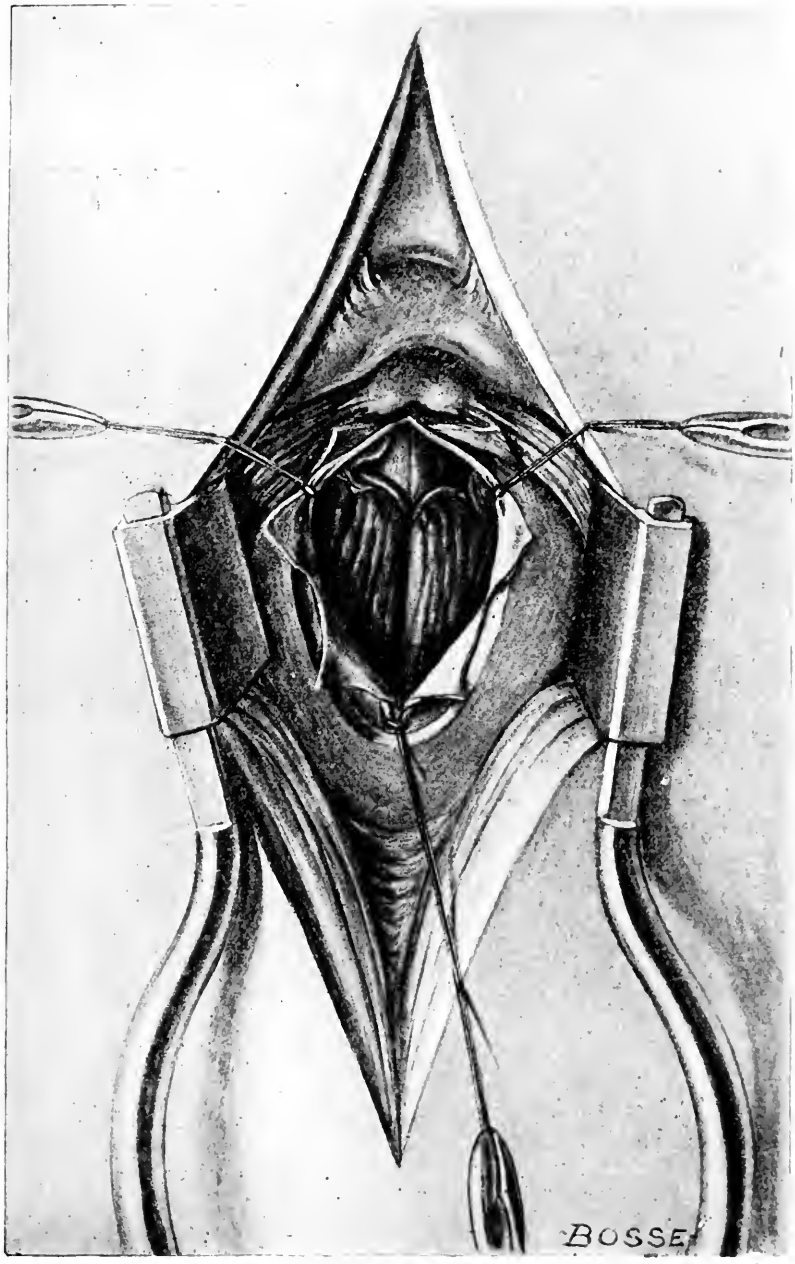


Fig. 11.—The sinuses have been ligated and the dura and arachnoid widely reflected. The two arteries shown curving around the poles of the cerebellum are the posterior inferior cerebellar. They seem to be in close relation with the medulla. They are not. It is impossible to represent, in an illustration, the real depth of the cistern at the bottom of which lies the medulla.

Fig. 12.—The instruments which will be found useful in performing this operation. It will be noted that the most of them are "stock" instruments, found everywhere, and do not require even mention.

Attention is called to the self-retaining retractor which is of great assistance in maintaining the wound fully exposed. Also, to a bone cutter, which has a reversible cutting end that may be used in four different positions. Further, to the cerebellar "spoons or pushers" with which the lobes of the cerebellum may be gently pushed upward and apart.

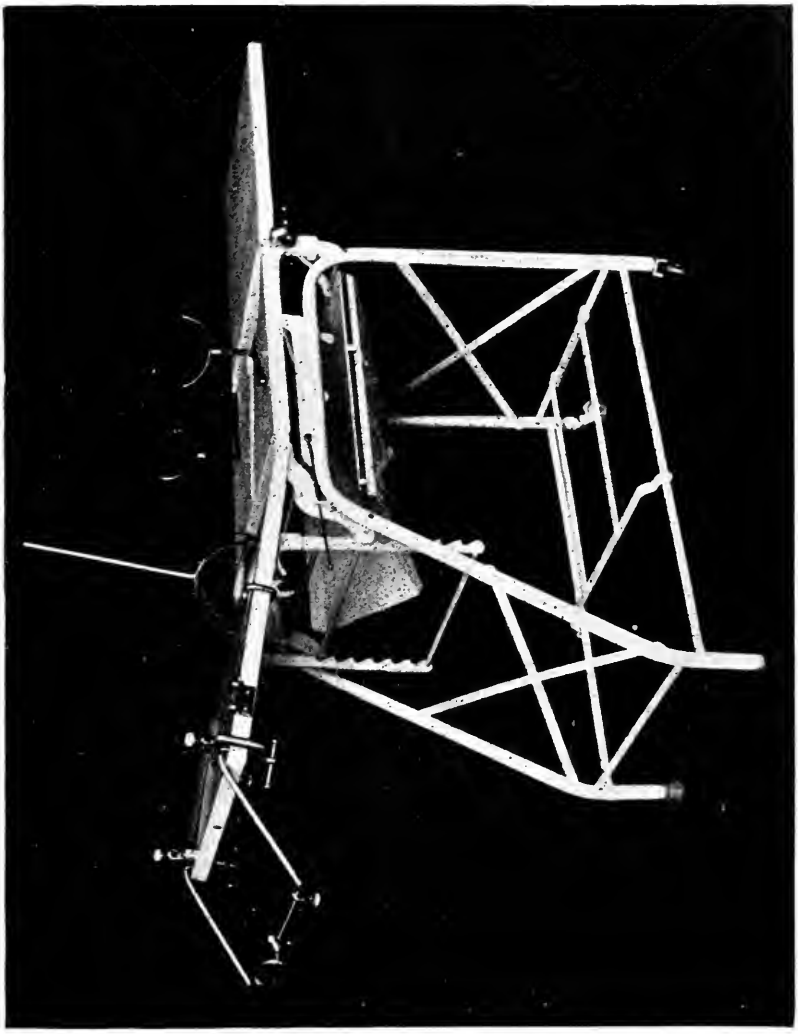


Fig. 13.—Detached portions of the head-rest shown.

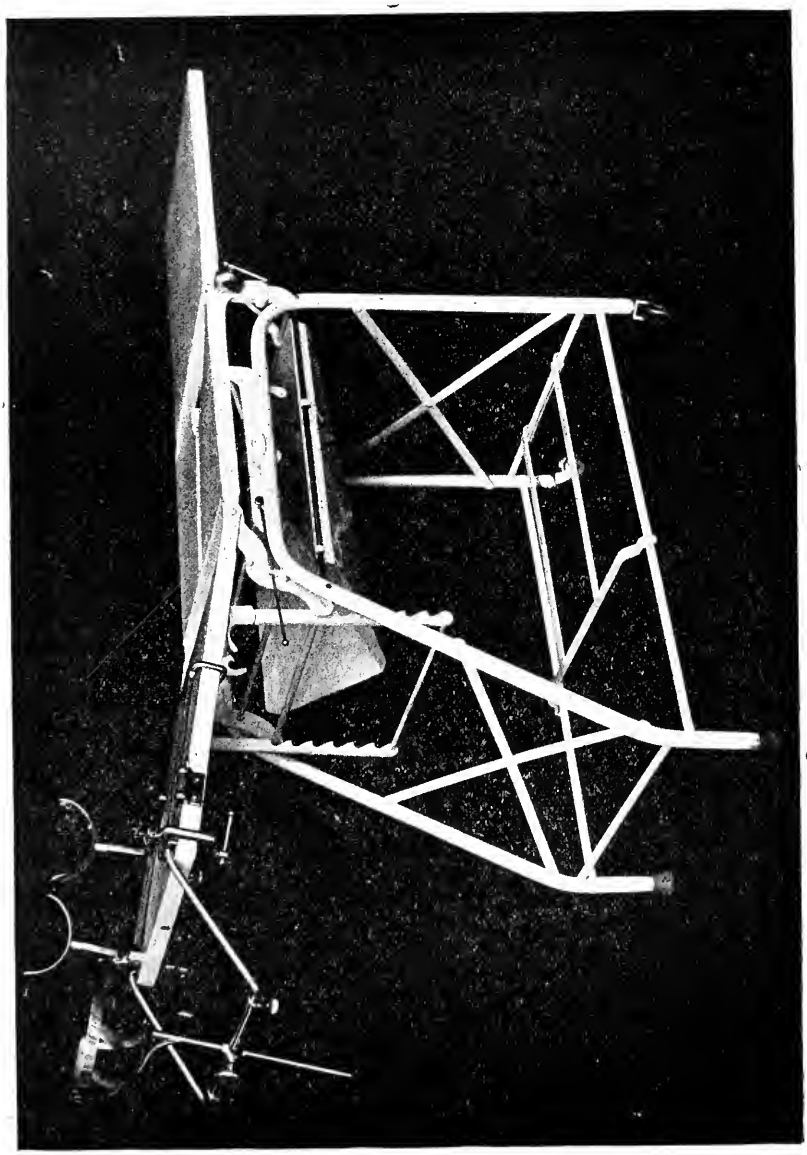


Fig. 14.—The head-rest assembled. The table should be horizontal. It was inclined by the photographer.

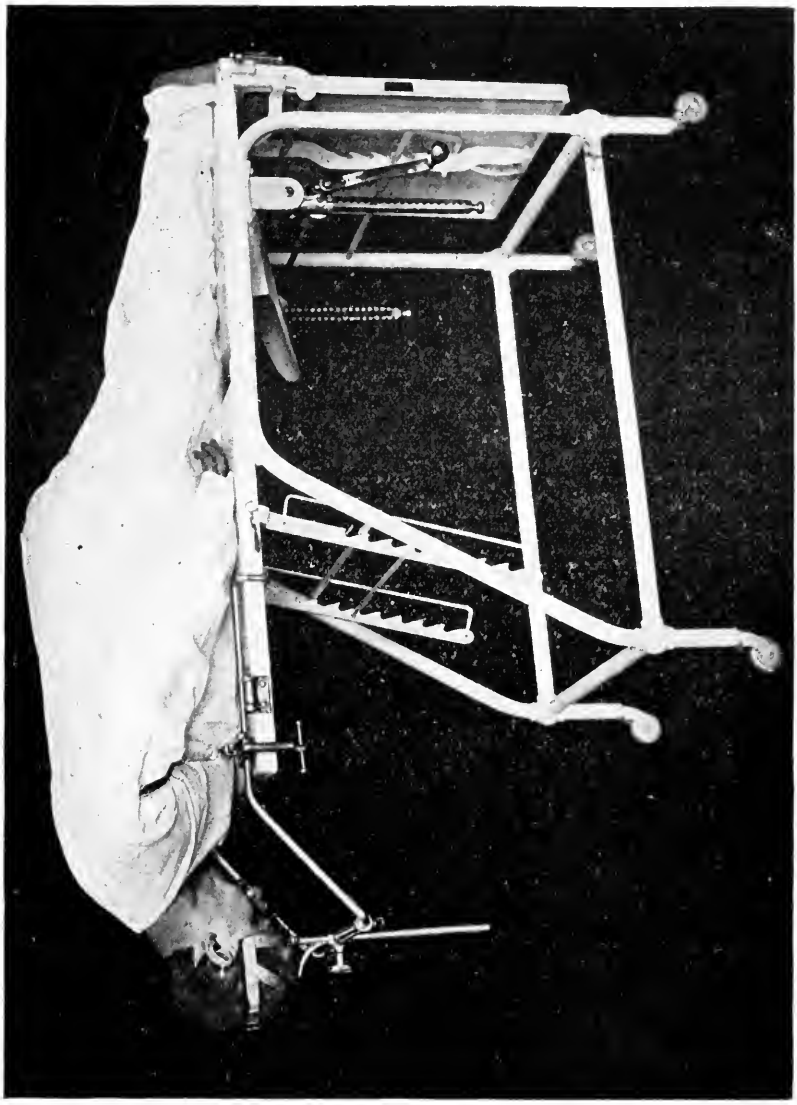


Fig. 15.—Shows the position of a patient with the head rest in use. In practice fixation of the head is secured by placing towels in the head piece until a tight fit is obtained for the head.

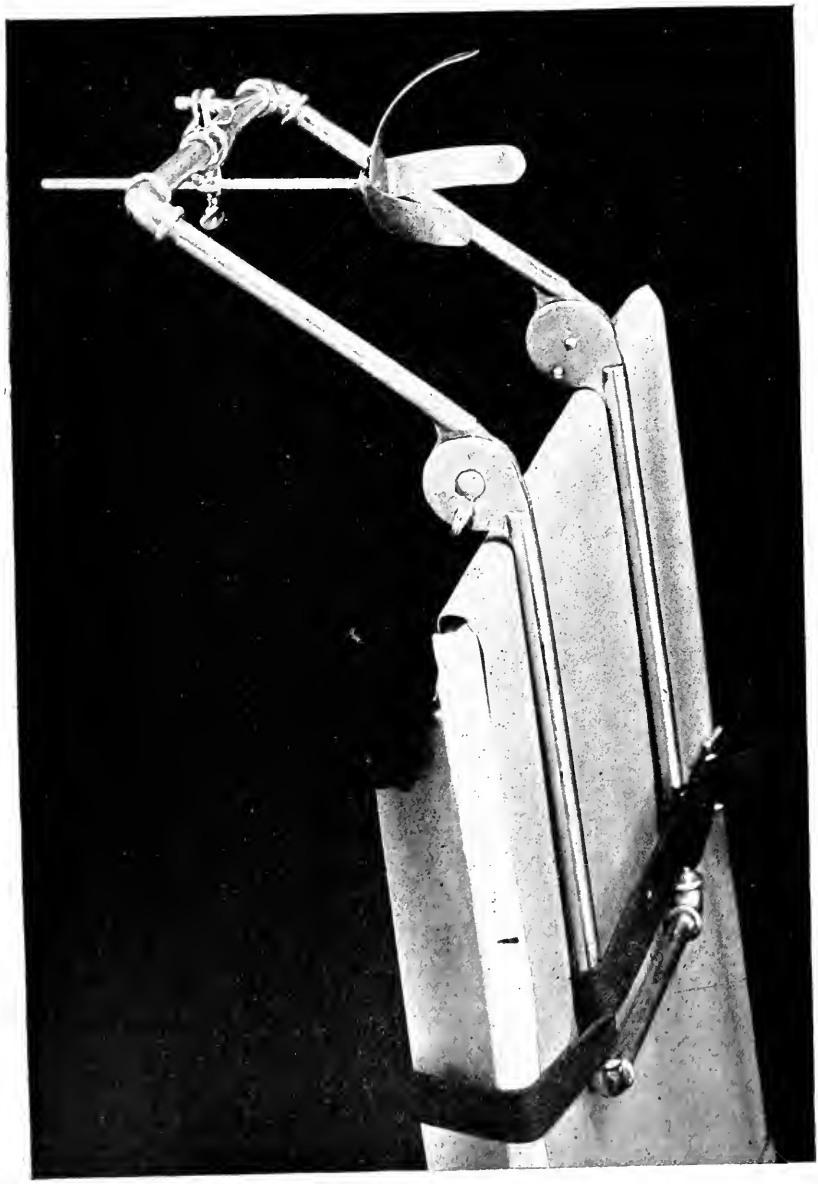


Fig. 16.—The author's head-rest attached to a common kitchen table by a webbing trunk strap.

cephalus. He states in a footnote that the normal pressure of the cerebro-spinal fluid, with the child in a horizontal position, was about 4 mm. of mercury.

The third case reported by Wynter was another laminectomy operation which was performed by Pierce Gould, on June 21, 1890. He cut down upon and divided the lamina of the first lumbar vertebra. The spinal canal was opened with a knife. A slight amount of fluid escaped, a drainage tube was inserted, the symptoms improved for a short time but the patient died. (Boy, 2 years old).

A fourth case in Wynter's report was a girl of 13 months in convulsions, semi-conscious, head retracted. April 15, 1891, a Southey's tube was introduced into the spinal canal in the lumbar region. Death followed in a few hours. Wynter remarks: "Though none of these cases were ultimately successful, no harm in any one resulted from the interference. In some there was temporary relief of the symptoms and the necropsy in each case showed ample reason for the fatal termination, either in general tuberculosis or failure in maintaining the drainage. Further experience will, no doubt, enable a better selection of cases to be made, and the treatment to be carried out more effectually.

Charles A. Morton (8) published a paper in 1891, upon "The Pathology of Tuberculous Meningitis with Reference to its Treatment by Tapping the Subarachnoid Space of the Spinal Cord." He says: "In view of the recent procedures of tapping the subarachnoid space of the spinal cord in cases of tuberculous meningitis, the pathology of the disease derives quite a new importance. The prospect of relief by surgical intervention in these cases depends on the relation which increased intraventricular pressure bears to other pathological changes and to the symptoms, and whether, from the conditions found after death, there seems a fair chance of our being able to relieve this pressure by tapping the subarachnoid space of the spinal cord." Morton's statements are introduced here to show that at this time he was thinking of the possibility of drainage in tubercular meningitis.

Victor Horsley (9), in December, 1890, in reference to the treatment of septic meningitis, proposed "Free drainage and disinfection, and the immediate consequences—relief." Further, "I have put down septic meningitis as a condition which might be improved by operation, such as free drainage and irrigation with warm disinfectants. I do not, however, believe that this course will always successfully rescue these otherwise hopeless cases. In fact, I have operated twice, once by way of exploration and once deliberately, but in both the moribund patients succumbed, after temporary though marked remission of their symptoms."

To Ballance (10) probably belongs the credit for first attacking meningitis by the occipital route. His statements made in 1897, "with regard to the operation of draining the posterior subarachnoid cistern, which had been described by Parkin in 1893, who had first performed it in 1891. The occipital bone was trephined close to the foramen magnum. One case died of hemorrhage from opening an abnormal sinus, which was an accessory lateral one situated lower than usual. Some cases had been opened on both sides of the middle line so as to prevent hernia of the cerebellum interfering with drainage."

While Ballance was undoubtedly the first surgeon to deliberately drain the occipital fassa for septic meningitis, his work was not made public until six years later, and to Alfred Parkin (11), who independently planned, executed and published the following operation, we should accord as much credit as if he had, in point of time, been the first to perform the operation. His case is as follows:

On April 9, 1893, Parkin operated upon a child 4½ years of age, who had

been sick for a month with meningitis. At the time of the operation there was Cheyne-Stokes respiration, swelling of the optic discs, transient rigidity of the limbs, unconsciousness almost amounting to coma. Pulse 150, and respirations 50. No anesthetic was required. "An incision was made below the superior curved line of the occipital bone and to right of the middle line. The flap was reflected and a circle of bone, three-fourths of an inch in diameter removed and the opening enlarged in a downward direction. The dura bulged very much, no pulsation was felt. It was opened and a little fluid escaped. On passing a curved probe along the under surface of the cerebellum and raising it slightly a very large quantity of clear cerebro-spinal fluid (about two or three ounces), gushed out; pressure was relieved and the brain pulsation returned. The Cheyne-Stokes rhythm ceased and the pulse became larger and regular, though still very quick. A very fine, almost capillary drainage tube was inserted beneath the dura mater and both it and the scalp wound were sewn up." In spite of free drainage up to the last, the child died sixteen hours later. Necropsy showed generalized tuberculosis of lungs, liver and meninges.

In this connection I wish to refer again to the work of Morton (12), whose operation for subarachnoid drainage has been referred to by later writers who give an erroneous idea of what Morton really intended to accomplish.

In order to make the matter clear, I will quote from Morton's papers on "The Opening Between the Fourth Ventricle and Subarachnoid Space and its Condition in Tuberculous Meningitis," and the "Best Point for Direct Drainage of the Ventricles."

Morton states that in 1891 he gave a paper on the pathology of tubercular meningitis, to which reference has already been made with regard to its treatment by tapping the subarachnoid space of the cord. Since then he has investigated the condition of the opening from the fourth ventricle into the posterior sabarachnoid space, in seven normal and seven cases of tubercular meningitis. It is evident that if the foramen of Magendie be closed, drainage of the ventricles of the brain by tapping the subarachnoid space is impossible. His examinations show that the foramen is never closed.

"Morton's method" for exposing the foramen of Magendie so as not to disturb its condition or relations consisted in first removing the skull-cap, the section being carried low down behind. Then a wedge-shaped piece is removed from the middle of the occipital bone, with its base uppermost and with the apex, almost an inch wide, forming the upper boundary of the foramen magnum. The removal of this wedge exposes the dura which is incised, the arachnoid is exposed and also incised. The fluid is gently sucked up with a sponge. "In this method there is no possibility of rupture of the pia mater closing in the ventricle."

In reference to the second point in his paper, "the drainage of the ventricles," he advises that they be drained "through a trephine opening, situated one and one-fourth to one and one-half inch behind and the same distance above the external meatus."

"Morton's method" then, in so far as it referred to the posterior subarachnoid space, was a method of exposing the foramen of Magendie, post mortem so as not to disturb its condition or relations. It had nothing to do with drainage of this space except to show that the foramen of Magendie was never closed. Therefore this space could be drained by way of the spinal subarachnoid space (already demonstrated by Wynter and Quincke), or through the lateral ventricles (previously advocated and performed by Bergmann and others).

Ord and Waterhouse (13) reported the recovery of a girl, 5 years of age, who was nearly moribund with "tubercular meningitis with increased intra-cra-

nal pressure, the stage of coma rapidly advancing and unless pressure was relieved, a fatal termination seemed inevitable." The operation was done under chloroform, October 26, 1893. "A curved incision, two-and-a-half inches long, over the left cerebellar fossa of the occipital bone, commencing below and behind the mastoid process, passing with its convexity upward and ending externally to the external occipital crest. Furious bleeding from the occipital artery. Some bleeding from a torn mastoid vein. Pericranium raised in a flap similar to the scalp flap. A three-quarter of an inch trephine hole was made midway between the external occipital crest and the mastoid process. The dura bulged tensely into the opening, there were no pulsations. The dura and the arachnoid were incised and some thirty drops of a slightly greenish serous fluid escaped. The cerebellum bulged into the trephine opening, fitting it tightly like a cork in a bottle. A probe was passed between the cerebellum and the arachnoid towards the falx cerebelli and some drachms of fluid escaped. A drainage tube was passed along the probe and left in position. Fluid slowly passed along it. The dura was sutured, the fragmented bone-button packed in the opening and the flap sutured with the drainage tube escaping through a stab-hole in its middle. Drainage continued until the end of the following month, when recovery was complete."

In this connection we should note that Fischer (14) refers to a case sent by Browning, of Brooklyn, to the Bushwick Hospital, in which an operation was performed at the occipital site.

After disappointments became numerous in the use of lumbar puncture as a therapeutical measure in meningitis, the introduction of various drugs into the subarachnoid space was attempted. Thus Rolgaus (15), in two cases of tubercular meningitis, injected into the "subdural" (?) cavity an emulsion of iodoform, but without result. Next Sokolov (16), reflecting upon the similarity between tubercular peritonitis and meningitis and, being familiar with the curative action of merely exposing the viscera to the atmospheric air in the former, attempted to carry out the analogy in the treatment of this form of meningitis. He trephined, aspirated the cerebro-spinal fluid through a lumbar puncture and then insufflated air into the subarachnoid space through the lumbar puncture. He did not state the results of this treatment.

A combination of craniotomy and lumbar puncture was performed by Graaenigo (17) in three cases of suppurative otitic leptomeningitis, with recovery in all.

Chipault (18), in 1894, advocated opening the "Sylvian lake" for drainage purposes in chronic hydrocephalus and tubercular meningitis.

Witzel (19), in September, 1897, operated upon a boy, 16 years old, who had a circumscribed phlegmonous meningitis following right ear disease, by widely removing the squamous portion of the temporal bone and introducing gauze drainage after ventricular puncture had been negative. The patient died after four months of illness, with generalized meningitis. He also reports a second case of fracture of the base of the skull, followed by basilar meningitis, that was cured by the temporal operation.

In January, 1899, Rolleson and Allingham (20) treated a case of cerebro-spinal meningitis in a man, 24 years of age, by performing a laminectomy of the seventh and eighth dorsal vertebra. Drainage was provided by a rubber tube which was removed on the ninth day. The patient recovered. They remark, "as far as we can find out the spinal dura has not been freely opened before in acute cerebro-spinal meningitis." Evidently they did not know of Wynter's case.

When we come to consider cerebro-spinal meningitis due to the extension from purulent foci within the skull, the role that suppurative ear disease plays is a very important one, and, while a consideration of this special aspect of the subject is foreign, at this time, to our purpose, yet to make the review more complete, these cases are introduced here.

To Macewen (21) we unhesitatingly give the credit for pioneer planning and operating in this class of meningeal inflammation. He gives a summary of twelve cases of infective, purulent lepto-meningitis, of which six were operated upon and recovered. Five of these were of the cerebral fossa and one of the cerebellar fossa. Six other cases, considered hopeless for operation, died. Of six other cases of cerebro-spinal lepto-meningitis, five were operated upon with one recovery.

Hinsberg (22) reported seven cures and five temporary improvements in meningitis of otitic origin, after drainage of the subarachnoid cavity.

In otological literature there are numerous similar cases on record in which a craniotomy in connection with the operation upon the diseased ear was performed to reach and drain a circumscribed or general purulent meningitis Lumbar puncture, as above noted, has been used for purposes of diagnosis and a possible therapeutic effect.

Friedrich (23), in cases of cerebro-spinal meningitis of otitic origin, advocated incision of the dura at the site of the infection and in addition performing a laminectomy. Held and Kopetzky (24) give the details of a remarkable case of purulent meningitis following a double otitis media in which pus was obtained from the subarachnoid space in the temporal region, through the enlarged mastoid wound, from the spinal space by lumbar puncture and from the lateral ventricle through puncture. The girl, 3 years of age, finally recovered after this widely disseminated infection in which were found streptococci and staphylococci, the latter predominating.

Kuemmel's (25) case, frequently quoted, is introduced here to further emphasize the possibility of recovery from a purulent meningitis after a double trephining of the occipital bone and the institution of drainage.

The patient was a man of 33 years, who sustained a fracture at the base of the skull. Lumbar puncture drew off 20 ccm. of purulent fluid under a pressure of 235 mm. of mercury without relief. After the occipital operation the patient began to improve and fully recovered in six weeks.

In 1906, Widal and Ramond (26) treated a case of cerebro-spinal meningitis by injecting through a lumbar puncture 5 cm. of a 1-100 solution of collargol in water. This was repeated a second time on the twenty-ninth day and the patient discharged cured on the thirty-sixth.

In April, 1907, the anti-meningitis serum, prepared at the Rockefeller Institute under the direction of Flexner, was used for the first time in treating epidemic cerebro-spinal meningitis in human beings, by Ladd, of Cleveland (Dunn) (27). This serum was introduced through a lumbar puncture made in the usual way.

Radman (28), in writing upon the surgical treatment of epidemic meningitis, advocates drainage of the fourth ventricle by an incision through the atlanto-occipital ligament. Fischer (29) reports a case of cerebro-spinal meningitis in an infant, 2 months old, in which recovery seemed to follow repeated irrigation of the lateral ventricles with a normal saline solution, combined with several lumbar punctures. Pollack (30), among some other cerebral lesions, recommends puncture of the brain in hydrocephalus after his method of introducing the needle through a small drill-hole in the skull. Bramann (31) has treated eighteen cases of hydrocephalus by puncture of the corpus callosum.

Brem and Zeiler (32) suggest the treatment of influenzal meningitis by permanent spinal drainage combined with the internal administration of hexamethylenamin.

Chapter II.—Anatomy and Physiology.

It is assumed that the reader is familiar with the fundamentals of brain and cord anatomy and physiology. Some parts, however, which are especially concerned in the problems before us, must be emphasized.

The Anatomy.

The Cerebro-spinal Cavity:—The cerebro-spinal space in the natural condition is a closed cavity, limited by bones and firm ligaments. It is incapable of expansion or distention except in the infant with open fontenelles and then only to a very slight amount.

The Brain and Cord:—The brain consists of two symmetrical hemispheres which are united over a restricted portion of their opposed surfaces by the wide colossal commissure above and the basal structures below. From the base of the brain fibers emerge, crowded together to form the crura cerebri and passing downward to become the pons, medulla and spinal cord. The cerebellum, beneath the hinder portion of the cerebrum and behind the pons and medulla is united to these parts by masses of fiber. All these last parts together constitute the brain.

The Ventricles of the Brain:—Within the substance of the cerebral hemispheres lies the spaces called the lateral ventricles, while between the hemispheres is another space, the third ventricle. The cavity of the former spaces is continued into that of the latter by two semilunar slit-like openings, the foramina of Monro. Between the cerebellum, pons and medulla lies another wide space, the fourth ventricle, which is united to the third ventricle by the aqueduct of Sylvius, a narrow tunnel through the hinder part of the cerebral crura.

While the cavity of the fourth ventricle is mostly walled in by the substance of the cerebellum, pons and medulla its posterior wall or "roof" is furnished by the pia lined by a thin membrane (representing the attenuated roof of the primitive brain), and termed the tela choroidea inferior. This "roof" is incomplete in its central portion near the lower part of the fourth ventricle, where an opening is found—the foramen of Magendie—by which communication is established between these spaces within the brain and those (to be subsequently mentioned) without. Further, narrow gaps are found in the lateral angles of the fourth ventricle which also open into the spaces without the brain. These are the foramina of Luschka, or Key, and of Retzius. In passing, we should mention that the fourth ventricle is continued throughout the spinal cord as a small central canal.

The Membranes of the Brain:—The dura. This is a dense membrane lining the interior of the skull—forming its internal periosteum—also extending to the bottom of the spinal canal—but not acting as a periosteum for the vertebrae. It smooths out inequalities of the cranium and effectually blocks up all points of exit therefrom. From the dura are derived two large incomplete diaphragms, the tentorium cerebelli and the falx cerebri. The former aids in forming a chamber within which lie the cerebellum, pons and medulla; the latter is suspended between the cerebral hemispheres. Along the margins of attachment of these partitions to the skull, and to each other and within the separated layers composing them are found large channels—the cranial sinuses—by which the venous blood is removed from the brain.

The Sinuses:—The cranial sinuses which enter at all into our consideration are the superior longitudinal, straight, lateral and occipital. The superior longitudinal sinus is especially important, not merely because of its size, but because of the connection with it of broad,irregular venous spaces called the parasinoidal sinuses or the lacunae laterales. These lateral lakes vary in width from one-half to one inch and into them, as well as into the sinus itself, open the cerebral veins and project the Pacchionian bodies (see latter).

The straight sinus receives the venous blood from the interior of the brain through the veins of Galen and opens, with the superior longitudinal into the confluence of sinuses from which pass off the lateral sinuses to the base of the skull where they are continuous with the internal jugular veins.

The occipital sinus is usually a small one in the falx cerebelli; it may be wanting entirely, may be double, and rarely may be of considerable size. It opens into the torcular above and near the margin of the foramen magnum bifurcates into two smaller venous tracts—the marginal sinuses—which course around the border of the foramen and empty into the lateral sinuses near their termination.

The Intradural or Subdural Space.—This is the very narrow interval left between the dura and the next membrane—arachnoid—it is a potential rather than a real "space" and harbors a very little cerebro-spinal fluid for purposes of prompting freedom of motion between the brain and the skull.

The Pia.—Immediately applied to the brain and the cord is the pia. A layer composed of blood vessels with only enough connective tissue to hold them in place. The pia follows all the irregularities of the surface of the brain, dipping into all the sulci and, further, projecting into the ventricular cavities as masses of capillary festoons—the choroid plexuses of the various ventricles. A word further about these vascular plexuses. While they seem to lie free within the ventricles, in reality they are excluded from these spaces by a layer of epithelium which represents the thinned-out wall of the primitive cerebral vesicles. This construction is important to remember when we come to take up the physiology of these structures.

Besides these vascular plexuses there are other capillary tufts—the Pacchionian bodies—outgrowths from the pia which look like small masses of granulation-tissue and consist of a spongy trabecular network covered by a membrane continuous with the arachnoid. They project through the inner layer of the dura into the superior longitudinal sinus and the lateral lakes; also, they are found in fewer numbers along the upper surface of the superior vermis of the cerebellum. The Pacchionian bodies, before the age of three years, are wanting or very small and rudimentary. From the age of ten and onward they increase in number and size.

The Arachnoid.—This is a delicate membrane interposed between the dura and pia but so intimately connected with the latter as to seem on casual examination a part of it, especially over the convexity of the brain. However, at the various sulci the arachnoid and pia are seen to be separated, for while the pia dips into the sulci the arachnoid bridges them over. It is at the base of the brain that the existence of two layers is most easily demonstrated. Here, while the pia closely follows all the irregularities of the parts, the arachnoid spreads from one elevation to another as a well-defined sheet. The spaces which are thus left between the pia and the arachnoid are narrow channels over the convexity of the brain, but at the base they are large spaces, and are given localizing names as cisterna basilis, cisterna pontis and cisterna cerebello-medullaris or magna.

These spaces communicate with each other in the freest manner possible; also, with the narrow channels over the convexity of the brain, and especially, at the cisterna magna not only with the wide subarachnoid space of the spinal canal but also with the fourth ventricle through the foramen of Magendie and the lateral foramina of Key and Retzius.

While there is more or less of a spongy tissue connecting the arachnoid with the pia over the convexity of the brain, still, as Cunningham (33) says: "In these (cisternae) there is no longer a close network; the trabeculae connecting the two membranes take the form of long filamentous, intersecting threads which traverse the spaces."

In this connection the attention of the reader is called to the foramen of Magendie. The text-books give no description of this opening that would enable one to form any conception of its shape or size; therefore I shall quote the correct picture given by Morton, (12) which also expresses my own conclusions in this particular.

"The opening is not accurately described as a foramen, in the sense of a small hole in tthe pia mater. The opening in the normal brain reaches from the cerebellum to the calamus scriptorius below, and is a broad slit or a large space. Sometimes a few delicate strands of membrane intersect one another across it but this is only occasionally seen."

Further, Lee (34) states that it is larger in infants than in the adult. Anatomically, there is no direct connection between the subdural, or better, the intradural space, and the subarachnoid or intra-arachnoid spaces, but the arachnoid tissue is so porous that, anatomically, physiologically and pathologically the intradural space may be considered as identical with the subarachnoid spaces.

The Blood Vessels.—The internal carotids and vertebrals furnish arterial blood to the base of the brain, which, through the mechanical construction of the circle of Willis is adequately distributed to the entire brain, both externally and internally. It is sufficient for our purpose to state that from the arteries, capillaries are formed, which again converging, unite to form gradually the various intra and extra-cerebral veins. The former are chiefly gathered up into the main trunks of the veins of Galen and these two empty into the straight sinus. The external veins course mostly over the convexity of the cerebrum and end in the superior longitudinal sinus or the lateral lakes opening into it. There are other venous connections with the sinuses, with each other, and with the veins of the diploe which we can disregard at this time.

The Lymphatics.—(Macewen, (21) page 38). "The lymphatics of the dura are abundant. There is besides, a wide-meshed capillary network with peculiar dilatations near the inner surface of the cranial dura. * * * The subdural space communicates with the lymphatics in the dura, while the latter communicate with the dural veins. The intra-cranial lymphatics have their origin in the cerebral pia matter and in the choroid plexuses. They pass out of the cranial cavity along with the internal cartoid and vetebral arteries and the internal jugular vein to the deep cervical glands. Others pass from the choroid plexuses of the lateral and third ventricles, coalescing into a lymphatic vessel which accompanies the veins of Galen."

Physiology.—The chief facts of interest to us respecting the circulation of the blood within the skull is that by various mechanical means the intermittent arterial flow is converted into a constant, steady stream. The details of this mechanism are well given by Macewen (21) (page 35), but do no require elaboration here. We, also, should note that there is always a positive pressure in

the cranial sinuses, amounting to a few millimeters of mercury. (Ballance (35). Of more importance to us is the cerebro-spinal fluid and its functions.

The cerebro-spinal fluid is secreted chiefly by the various choroidal plexuses. The current sets from the lateral ventricles through the foramina of Monro into the third ventricle and thence by the aqueduct of Sylvius into the fourth ventricle and from here it freely escapes into the cisterna magna and pontis through the large foramen of Magendie and the smaller foramina of Key and Retzius. From the cisterna magna the fluid passes slightly into the subarachnoid space of the spinal cord but mostly streams upward through the other cisterns and over the convexity of the brain into the cerebral veins and sinuses. "This direct passage of the cerebro-spinal fluid into the veins is one of the best established facts in the physiology of cerebral circulation, especially by way of the Pacchionian bodies" (Archibald (36).

The pressure of the cerebro-spinal fluid in the subarachnoid spaces always exceeds by a few millimeters of mercury the cerebral venous pressure; therefore the flow of fluid is from the subarachnoid space into the venous system. As the specific gravity of the cerebro-spinal fluid is less than that of the blood, any flow determined by osmosis would be, in the main, in the same direction. (Ballance).

The quantity of the cerebro-spinal varies with sex and age and never exceeds 200 ccm. (Gray 37), or 60 to 80 ccm. (Howell 38). It may, however, under the stimulation of injury and disease, be secreted in immense quantities. Giss (39) reports a loss of 30 liters in 37 days. Thompson, (quoted by Archibald), a loss of 500 ccm. in 24 hours.

Another function of the cerebro-spinal fluid is to form a "hydraulic buffer" (Ballance) between the cranium and the brain. As to its action in "supporting the weight of the brain" this is very slight, if any. Any one who has made and studied sections of the head and neck and the relations of the brain and cord to the surrounding parts will agree with Ballance that "the weight of the brain is not wholly, or even to any considerable extent supported by hydrostatic pressure."

The function of the Pacchionian bodies is so interesting and important in this study, that further notice is taken of it. Kocher (40) states it as follows:

With the expansion of the arteries at the base of the brain at each systole of the heart, the Pacchionian bodies and the cerebral veins are compressed and empty their contained cerebro-spinal fluid through the thin layer of the dura into the sinuses. This current is also aided, during the diastole of the heart, when the arteries again contract, by the expansion of the Pacchionian bodies aspirating the cerebro-spinal fluid from the subdural and subarachnoid spaces. The cerebro-spinal fluid does not leave the skull by the lymphatics unless there is venous obstruction raising the pressure within the sinuses. (Archibald).

The chief function, then, of the cerebro-spinal fluid is to maintain at a constant level the intra-cranial pressure, which tends to vary with cardiac action, respiratory rhythm and changes in bodily position.

In infants, until the closure of the fontenelles and while the Pacchionian bodies are rudimentary and their function unimportant, the normal intra-cranial pressure is maintained and the circulation of the cerebro-spinal fluid assisted by the alternate expansion and retraction of the fontenelles with arterial pulsation and respiratory movement.

Chapter III.—Operative Treatment of Meningitis.

A. Foreword. B. Critical Review of Past Operations. C. Symptoms Which Furnish a Guide for Operation. D. What May an Operation Promise?

E. When Should Operation be Undertaken? (a) To Save Life; (b) To Prevent Complications. F. Where should the Operation be Performed?

A. Foreword.—The historical review just given, while not exhaustive, is sufficiently comprehensive to give us a correct picture of meningitis treated by surgical measures. There is yet no medical treatment for this disease. What a picture it is of hopelessness, despair and death! The only ray of light cast on this dark field is given by the serum treatment of one particular form of this disease. But the mortality remaining after the use of this remedy is still sufficiently appalling, that were it in any other disease, it would fill us with consternation. While in the other types of the disease death is the universal outcome, we have come to accept meningitis as a fatal disease. The facts of the past justify this conclusion. If, perchance, recovery did occur, how often would death have been preferable. A recovery with reason dethroned or weakened, with epileptic fits or other deplorable nerve lesions was more to be regretted than death. Is there, then, no hope? Must we fold our hands and let these patients die under stress of the greatest suffering?

We claim there is hope. We claim that if this disease is treated with common sense along lines of ordinary surgical technic used in other suppurative conditions, a fair percentage of these otherwise hopeless cases will recover.

What are the facts in the case? 1. That medical treatment is unavailing. 2. That surgical aid in every case reported has been deferred until the patints were moribund.

Ante-mortem surgery has never given, and can never give, "good results." When the dictum is accepted in meningitis, as it is in appendicitis, mastoiditis and other suppurative diseases, that the only treatment is surgery, and that utilized at the earliest possible moment after the diagnosis is made, then, and only then, will the high mortality statistics of meningitis begin to fall.

The same fight is on here that was waged by surgeons with their medical confreres in the past over similar forms of suppurative disease. Take appendicitis. We know the steps in that contest. 1. Medical measures alone; 2. operating in moribund cases; 3. in abscess cases; 4. in the acute attack before sepsis has had a chance to do any damage; 5. in the interval. How much fine hair-splitting was indulged in, in the past, as to when a case of appendicitis ceased to be a medical and became a surgical one.

What protests went up when Deaver announced, and adhered to it, that appendicitis is always a surgical condition and demands surgical treatment just as soon as the diagnosis is made.

Other similar affections, now relinquished to the surgeon, without a question, have had to go through the same cycle of evolution, and, I suppose, meningitis will prove no exception.

3. While admitting the futility of medical treatment and the desirability of surgical interference in the past, there has been no concerted action among surgeons themselves to develop an operation that would commend itself as reasonable, safe, and curative. The operations suggested and performed have had to do with some particular form of the disease and at the terminal stage of its course. Furthermore, they have been futile if simple, or so severe in their technic as to be contra-indicated.

4. The indications as to the proper time for surgical interference have not been clearly formulated. This has been due to the uncertainties of a positive diagnosis at an early stage in the disease; also, to the confusion arising from the multiplicity of types of meningitis, each one described as an entity based upon

its etiology and post-mortem findings and none of them considered from the standpoint of possible surgical opportunities.

B. Critical Review of Past Operations.

Ventricular Puncture.—Ventricular puncture after the method of Kocher, Keen (41) or Pollock, through the cerebral cortex, or of Bramann, through the corpus callosum, is a procedure only to be utilized in cases of chronic hydrocephalus or in non-pyogenic serous effusions of the brain.

"In infected cases with a beginning external meningitis there is always a certain risk of innoculating an uninfected ventricle." Cushing (42). In addition there is a real danger in infecting the cerebral cortex and more especially the meninges. Further, hemorrhage from a cerebral vein or from an injury to the choroid plexus is a not unlikely possibility. If ventricular puncture is performed for the purpose of draining a purulent effusion at the base of the brain, one only has to consider the anatomy of the parts involved to perceive at once the futility of such a procedure. Bruce Clark (43) states that he does not "think it possible to relieve purulent basic meningitis by tapping the ventricles." That the operation in itself is a serious one is conceded by all. Frazier (44) says, "puncture of the ventricles (lateral) is unfortunately an operation of unusual gravity and the danger attending it is so great in comparison to the possible benefit as to make it a procedure of questionable propriety." If that is true of the operation in non-infected conditions, how much more is it objectionable in septic effusions at the base of the brain to which is added the severe constitutional effects of such disease.

It is plain then, that ventricular puncture, as a therapeutic measure in the treatment of meningitis has no logical or clinical justification. It cannot drain pus. It may not relieve pressure. It adds a positive danger of infection of ventricles, cortex and meninges. Troublesome hemorrhage may be produced. The operation is a very serious one.

Lumbar Puncture.—The value of lumbar puncture lies in the positive diagnostic findings furnished by the cerebro-spinal fluid withdrawn by the puncture. As a means of diagnosis then, it is invaluable.

As a therapeutic measure it is inefficient except in the single condition of serous, non-purulent, meningitis. In all purulent conditions it has no curative powers. At its best, in epidemic cerebro-spinal meningitis lumbar puncture has a mortality of 66.6 per cent (Sladen (45) in cases treated at the Johns Hopkins Hospital, before the introduction of the anti-meningococcus serum. In the frankly purulent infections lumbar puncture never cures. Archibald says in reference to it, "that value of lumbar puncture is therefore definitely proved to be minimal," also that, "lumbar puncture, while of temporary value, fails to save life;" further, "if a block has occurred at the foramen magnum, lumbar puncture only adds to its intensity" and "in ventricular obstruction lumbar puncture ceases to be helpful." Cushing also warns against the danger of too quickly withdrawing the column of cerebro-spinal fluid and causing a plugging of the foramen magnum by the brain stem. Frasier says, "the sudden disturbance of pressure is no doubt responsible for a large majority of the fatalities. * * * Furbinger, who is very much opposed to this practice, attributes the deaths to pressure exerted upon the bulb by the arrest of cerebro-spinal fluid from the ventricles at the foramen of Magendie."

Lumbar puncture then, as a therapeutic agent, is a failure. It possesses elements of danger, when repeatedly used, which disqualify it. Not only does it fail to reduce mortality, but its repeated use is attended with grave complications.

Lumbar Puncture in Combination.—(a) With craniotomy for through and through irrigation; (b) for the injection of various curative agents (except serum) into the spinal canal has been tried and discarded as "impossible of performance" (Cushing) and incapable of curing the disease and positively dangerous. The single case of recovery after the injection of collargol was a type of serous meningitis in which recovery might have followed the puncture alone.

Laminectomy.—There is more to be said in its favor as a curative measure than for lumbar puncture. Yet the danger of "corking up the foramen magnum by the brain stem," the failure to secure adequate and sustained drainage, the severity of the operation itself all weigh against it, if there is any other proposition which will attain the desired ends without these drawbacks.

Serum Therapy.—This is limited to the treatment of cerebro-spinal fever caused by the diplococcus intracellularis of Weichselbaum. (Flexner 46). It affects no other form of meningitis. In its particular field it has lowered a previous mortality of 72 to 95 per cent down to from 20 to 43 per cent (De Meric 47) or of a mortality of 66.6 per cent to 17.4 per cent. (Sladen).

There is no longer any question but that Flexner's serum is a potent agent in reducing the mortality of epidemic meningitis from a previous figure of about 75 per cent down to one around 25 per cent. The beneficial effects of the serum are more manifest if given before the fourth day of the disease and between the ages of 2 to 10 years. If given later than the fourth day or to patients younger than two years or older than ten, the mortality rate quickly rises.

In all other forms of infection the serum has no curative action whatever. At the best then, in a series of selected cases, (time and age most favorable), the mortality rate is 13.1 per cent while the highest is 43 per cent. (Dunn).

The average we can take at 25 per cent. (it is above this*). What is to be done regarding this remaining fourth of epidemic meningitis cases and practically the total number of other forms of infectious meningitis?

While we believe in and advocate the use of the serum in the diplococcic form of meningitis, it must be remembered that there will be failures in its use amounting, as shown, from 10 per cent to 25 per cent of the cases. In addition, the serum in itself is not altogether harmless as shown by the cases reported by Hutinel, (48) four in number, in which death followed the second injection of the antimeningococcic serum. The danger of death is greater should the infection be the tubercular bacillus.

Ryfkogel (40) reported a case of coccidioidal meningitis in which death followed a second injection of the Flexner's serum. Netter, Courtois-Suffit and Dubosc (50) advise caution in the use of the serum, in too large doses or too frequently repeated.

The remaining varieties of cranial operations will be discussed in connection with the operative procedure suggested in this paper.

C. Symptoms Which Furnish a Guide for Operation.

The subject of meningitis is obscured by describing as separate entities various forms of meningeal inflammation. From an etiological and pathological

*Sophian's (62) latest figures based upon the serum treatment of meningitis in the late Texas epidemic give the general mortality as 25 per cent. in a total of 185 cases. "Excluding the apparently hopeless cases in which the patients died in from a few minutes to twenty-four hours after admission, the mortality was about 10 per cent. In children the mortality after the exclusion of apparently hopeless cases was roughly about 5 or 6 per cent." This again gives the very best showing for the serum therapy after all the "hopeless" cases have been weeded out. As soon as one starts this weeding-out process one can get almost any kind of mortality statistics.

NOTE:—No attempt has been made to ascertain all the disadvanages and dangers attending the use of the serum. We have given it all the credit claimed by its advocates and merely cite the above to show that its use may not only fail to do good in meningococcic meningitis but also may prove dangerous in this and other forms.

standpoint, this is correct. But from a surgical point of view, such multiplicity of types only confuses the observer and serves no helpful purpose. He loses sight of these fundamental facts:

1. That all forms of meningitis are essentially septic in nature except the toxic, and this may become septic.

2. That all forms of meningitis kill, some only 10 per cent., others 100 per cent.

3. That death is caused in the last analysis by increased intracranial pressure progressing to such a point as to finally shut the blood off from the "vital centers."

4. That death can only be averted by furnishing good blood to the "vital centers" by removal of the intra-cranial pressure. Other appropriate and secondary measures will contribute to but cannot take the place of this paramount requirement. Irrespective, then, of the particular type of meningitis, of what has or has not been done before for therapeutic purposes, the indication for surgical action rests upon an unfailing chain of symptoms that, in practice at the bedside, can be absolutely determined with accuracy.

These early symptoms are: 1. A rising blood-pressure; determined by the cardiac sphygmomanometer. 2. Edema of papillae. (Not "choked disc," which is so late a symptom that it ceases to have any value). 3. Absence of carbohydrates from the cerebro-spinal fluid. (Obtained by lumbar puncture). 4. An irritable or clouding sensorium.

In addition, there may be present: A vagus pulse; respirations, irregular in depth and rate.

With these pathognomonic symptoms present, no possible consideration can justify delay.

The nearer a patient is to bulbar paralysis the less likely that any operation will save life. Recovery from such compression may not be at all or slowly.

(Archibald.)

D. What May an Operation Promise.

While we concede that lumbar puncture may be all that is necessary in toxic meningitis, that by serum therapy epidemic meningitis has been rendered two-thirds less fatal than formerly, yet, for the remaining fourth of those sick with this form of meningitis and for all other forms of septic meningitis death is the inevitable outcome.

Further, while surgical procedures of the past have saved but a lamentably few cases, yet we must remember that surgery has invariably been resorted to as a last hope; that the patients were in the terminal stages of cerebral compression and also suffering from profound systemic poisoning, and that, as a rule, the operations performed upon the skull at a distance from or near to the seat of the trouble have all been, in themselves, of a very severe character.

We can readily understand, then, why surgery has thus far been unable to show any considerable number of victories.

Can Surgery Relieve the Compression?

The evidence furnished by all cases operated upon show conclusively that pressure has been relieved at least for the moment. Why, then, has death so

NOTE:—The facts of cerebral compression as determined by experiment and shown in disease of the meninges have been given in detail by my co-worker on this subject, Dr. Kopetzky; (see Laryngoscope, June, 1912). I should like to state, however, that we have repeated the experiments of Cushing in producing intra-cranial pressure, have demonstrated the accuracy of his observations, and the correctness of his conclusions, and, in addition, have produced in dogs, acute, septic meningitis and recorded the various changes resulting from such infection as observed through a glass window in the skull and by other methods of reducing physical phenomena to graphic record. The results of our experimental work demonstrate that intra-cranial pressure is the important feature of meningeal inflammation.

invariably followed? 1. Because such relief has not been furnished at an early stage. 2. Because such relief has not been of sufficiently long duration. 3. Because, in addition to the evil effects of compression of the brain, constitutional septic conditions existed that continued to act after operative relief to cause death. 4. Because the meningeal infection had become so diffused and the products of such infection so organized as to make it impossible for Nature to further resist the progress of the disease or remove the effects of the inflammation.

All evidence warrants the conclusion that surgery has relieved all cases and has saved some. And it has succeeded in doing this in spite of the operation being performed at the very last, usually at some place unrelated to the seat of the disease and more frequently by a method of unnecessary severity of execution.

E. When Should Operation Be Undertaken.

a. To Save Life.—All the evidence, experimental ante- and post-mortem show conclusively that an early operation is absolutely necessary. Kocher advises operation in every case of cerebral lesion when there is only a suspicion of pressure. "It can no longer be doubted that in some cases of suppurative meningitis recovery may be brought about by active intervention. We are no longer justified in regarding such cases as hopelessly lost, and in remaining with folded hands. Rather must we attempt to save them by doing the utmost within our power." Hinsberg (51).

"The reproach of such a death rate (in septic meningitis) will not be removed by refusal to operate, but by an early and universal recognition of the futility of the treatment by drugs" (Horsley). "All forms of meningitis, if unrelieved by art, tend to cause death." (Ballance).

The otologists as a class have recognized the importance of early operations in septic meningitis of otitic origin. Their teaching is to follow up the disease until all parts affected have been brought into the field of surgical relief. (Kopetzky.)

b. To Prevent Complications.—In serum treated cases complications attributable to the disease (epidemic cerebro-spinal meningitis) arose in 43.5 per cent. In non-serum cases these were 63.6 per cent of about the same character. (Sladen).

"Early recognition, followed by operation, have in certain cases checked the disease (infective meningitis), and as our experience ripens concerning them, the results will doubtless be improved." (Macewen.)

"Energetic prophylactic decompression would reduce to a minimum such disastrous results as optic neuritis, deafness, oculo-motor disturbances, and some psychopathies, all of which are due to the mechanical effects of the excessive intra-cranial pressure." (Hultgen 52). "Probably, in some cases, the removal of fluid under pressure from the intra-dural spaces will prevent the occurrence of suppurative meningitis" (Ballance). Cushing states that the cause of death is "purely mechanical, and is due to intra-cranial pressure. Also that meningitis is a self-limited disease, and if pressure can be relieved and time gained the inflammation will subside and the exudate will be reabsorbed."

"While unconsciousness is a forerunner of paralysis of the vital centers, it should not be waited for." (Archibald).

If we have shown that operation is justifiable, that it must be invoked early, the next question is:

F. Where Should the Operation be Performed.

Clearly not at the far end of a long, narrow canal as in lumbar puncture or

laminectomy for the reasons already adduced, which are, impossibility of adequate or sustained drainage and the inherent dangers of the procedure itself. Certainly not by a more dangerous and less efficient means, as ventricular puncture. But by an operation which will positively tap the *fons et origo* of the trouble at the base of the brain, namely, the cisterna magna. Ballance was probably the first to attack meningitis by the sub-occipital route. He was followed by Parkin, and Ord and Waterhouse. But the operations performed by them and by others who followed them have not opened the cistern easily and satisfactorily, they have all been attended with many difficulties and grave objections, as will be shown later.

Anatomy shows us that this is the largest subarachnoid space, that it is in the "freest communication" with the other spaces without the brain and cord and especially with the ventricular cavities within the brain through a foramen always large and seldom if ever closed.

Chipault (53) states that he has found only two cases on record in which a communication between the fourth ventricle and the subarachnoid space did not exist, one case reported by Quincke, of chronic meningitis, and one by Keen, of unilateral hydrocephalus.

Anatomy further shows us that the cisterna magna is located close to the lower portion of the occipital bone in a position favorable for operative attack.

Physiology demonstrates that the course of the cerebro-spinal fluid is from the ventricles into the cisterna magna. Normally the current is of moderate force and the fluid in small amount. But, comes infection, the increase in amount becomes enormous and the rate of flow must be proportionately increased, so that we can imagine the fluid streaming into this space like a turbulent river into a broad lake.

Pathology offers its evidence that in this region the fires of meningitis burn fiercest and the debris of such inflammatory conflagration are in greatest evidence.

Campbell and Rowland (54) comment on the pathological findings in 206 cases of pneumonic meningitis, "the base of the brain shows the most marked lesions. The exudate is almost entirely confined to the base of the brain." Koplick, (55) in basic meningitis, found "lymph at the base of the brain and cord." "Thickening of pia and arachnoid at base of brain, some adhesions between cerebellum and medulla." Barlow and Lees, quoted by Koplick, state that the "primary seat of the inflammation is the region where the brain and cord meet and where the cerebellum overlaps the medulla." "Adhesions may result in blocking the foramen of Magendie."

Adami and Nicholls (56) "the exudate tends to collect at the base of the brain and along the posterior aspect of the cord. It is largely serous, but may be turbid. The exudate is found chiefly along the vessels and fills up the cisterns at the base of the brain."

Therefore, the conclusion is inevitable that the cisterna magna is the best and only reasonable place for surgical interference in meningitis.

Chapter IV.—Surgical Anatomy of the Sub-Occipital Regions.

1. The skin and subcutaneous fascia require no mention. They are divided by a vertical incision immediately in the median line.

2. The muscles lie in paired groups on either side of the middle line; consequently they are not divided in the operation, but merely retraction to one side. They are the trapezius, complexus and the rectus capitis posticus minor.

3. The nerves do not appear at all. The only one which might enter the

field is the third occipital. As this is a purely sensory nerve, its division could only cause temporary numbness of the back part of the scalp.

4. The vessels are the only structures of the soft parts which deserve more than a passing remark. They are paired arteries and veins which are connected more or less freely by anastomic vessels which cross the line of the incision. When divided they bleed, but the hemorrhage is easily checked by clamps.

5. The pericranium is easily stripped from the bone, and with it the portions of the origins of the muscles near the middle line. It is also directly continuous with the atlanto-occipital ligament at the margin of the foramen magnum.

6. A small median emissary vein may exist at about the middle of the occipital crest. It is not constant. If present it may be plugged by a piece of wood or by boring into the opening with the tip of an artery clamp.

7. The skull deserves a longer notice. There is a great difference in the thickness of the skulls in this region in adults. Of course in children it is very thin, with a slight thickening along the occipital crest. In both classes the thickest portion of the bone is at the occipital crest. On either side of the crest the bone becomes thinner, to again thicken up about the foramen magnum. In all cases it should be perforated with due caution.

8. The dura is thick and firm. It is readily separated from the bone. At the foramen magnum it is the most closely attached, but can be readily detached from the bone without difficulty. It is continuous here with the pericranium.

9. The occipital sinus "is the smallest of all the sinuses, about 2 mm. in diameter. It is composed of two parts: a vertical part which descends in the falx cerebelli; a horizontal or anterior part, also called the sinus marginalis, which courses around the margin of the foramen magnum and opens into the lateral sinus." (Poier and Charpy, 57). They also give the following figures as to its variation: 5 times out of 50 it was very large (Dumont); absent twice in 44 cases; 10 times the marginal branch was absent; in 9 times there were two sinuses; in 33 times there was one trunk unpaired and median, bifurcating at its lower part."

10. The arachnoid presents in this region as a membrane of considerable strength and individuality. It is closely applied to the dura and consequently separated by a wide space from the pia beneath, over an interval corresponding to the apices of the cerebellar lobes and the sides of the medulla.

11. The incisura cerebelli posterior. This gap between the cerebellar lobes is bounded below and in front by the medulla.

12. Covering the surface of the parts will be seen the vascular pia, with the two posterior inferior cerebellar arteries (from the vertebrals) standing out conspicuously as they wind around the apices of the cerebellar lobes from its lower surface into the incisura cerebelli.

13. If the cerebellar lobes are gently raised and separated there will be seen the opening, usually vertically placed and with irregular borders, the foramen of Magendie, which exists in the pia extending from the cerebellum to the medulla.

14. As to the cerebellum and medulla, our surgical and anatomical investigations do not carry us that far at this time.

Chapter V.—Drainage of the Cisterna Magna for Meningitis.

A. Purposes of the Operation. B. Steps of the Operation. C. Difficulties of the Operation. D. Advantages of the Operation. E. Secondary Remedial Measures. F. Other Conditions in Which This Operation May be Indicated. G. Instruments and Appliances.

A. Purpose of the Operation.

1. To open the cisterna magna, relieve intra-cranial pressure, and restore the normal supply of "good blood" to the "vital centers."

2. To do this without danger of cerebellar hernia or of "corking up the foramen magnum" by downward displacement of the brain stem.

3. To prevent shock and possible death of the patient from too sudden escape of the cerebro-spinal fluid.

4. To provide for free and continuous drainage of the infected cerebro-spinal fluid and thus enable Nature to effect a cure of the disease.

5. To afford inspection of the foramen of Magendie, and if it be closed, reopening it.

6. To forestall possible complications, especially hydrocephalus. If hydrocephalus is present, its relief and cure is possible by this operation.

7. To accomplish these desirable ends by an operation of the simplest technic, in the minimum of time, with the least shock.

B. Steps of the Operation.

The scalp has been shaved and the usual preoperative measures taken to secure and maintain absolute asepsis.

The proper position upon the operating table is maintained by the special head-rest (see figure) or by a competent, strong assistant, and sand bags.*

The anesthetic (ether) is administered through nasal tubes beneath the sterile sheet covering the patient.

The incision is in the middle line from the occipital protuberance to the spinous process of the axis and carried down to the occipital bone and posterior arch of the atlas.

Hemorrhage, slight and easily arrested, is checked by mosquito clamps and the vessels ligated.

The periosteum is now stripped from the occipital bone, taking with it the inner portions of the origin of the attached muscles, and the occipital bone is bared for about a distance of one and one-half to two inches vertically and an inch transversely, at the foramen magnum, less above (see illustration.)

The posterior arch of the atlas does not require baring.

An emissary vein may be encountered in the mid-line. It is not constant. If present it may be plugged by a wooden toothpick or by boring into it with an artery clamp.

The self-retaining retractor is now introduced. There are two sizes of detachable blades provided, one for adults and the other for children.

The De Vilbiss trephine (three-eights of an inch) is applied in the mid-line and about one inch from the margin of the foramen magnum and the button of bone removed. With the special dural separations the dura is loosened from the bone and the De Vilbiss bone-cutter used to make two lines of incisions or grooves through the bone into the foramen magnum. The dural separator must be constantly used to detach the dura from the bone in advance of the bone-cutter. For this reason these separators are made in two sizes and with a narrow shank to easily pass through the groove in the bone.

The wedge of bone, cut loose, is about half an inch wide at the foramen magnum and a little less at the upper border. Of course the size may vary for each individual and according to complications. The detachment of the bone-button is carefully completed and it is removed.

The dura presents, probably under pressure, bulging into the bone gap.

* A blood-pressure apparatus is attached to the patient and an assistant detailed to carefully watch this and the pulse and respirations and announce their variations.

The occipital sinus (or sinuses) will be seen, if present, showing a blue color through the dura. If the sinus is double, the dura should be incised between them. If single, it should be tied at the upper part and just beyond its bifurication into the marginal sinuses. The special, full-curved, right-angled dural needles, right and left, are provided for this purpose.

Caution.—In dividing the dura, first make a very tiny incision into it, using the fine curved bistoury for this purpose.* This is necessary, for should the arachnoid be so closely applied to the dura as to be divided with it we need to prevent a too sudden escape of the cerebro-spinal fluid. Should it be found that the dura has been severed alone the incision in it should be carried up and down to the limits of the opening in the bone.

The arachnoid will now bulge into the field, unless it has been divided with the dura. The amount of its bulging will give some idea of the degree of intracranial pressure. The arachnoid is slightly nicked in the middle line and the cerebro-spinal fluid allowed to escape, a specimen being taken for laboratory examination. While it escapes slowly a careful watch is maintained upon the blood-pressure, pulse and respirations by one especially detailed for this purpose. Syncope may be prevented or lessened by arresting for a moment the flow of the fluid by gentle pressure of the finger.

As soon as the excess of fluid has escaped, open the arachnoid for the full extent of the dural opening. The condition in the cerebello-medullary angle should be very carefully investigated. Should there be an exudate about the parts the lobes of the cerebellum should be raised and separated by the "pushers" provided for this purpose and the patency of the foramen of Magendie assured. It may be necessary to enlarge the opening in the occipital bone. This is easily done by the ordinary rongeurs or by the bone punch devised by the writer. A small wick of rubber or gutta-percha tissue is placed within the margins of the dura and arachnoid and left protruding from the wound. The muscles are replaced and held together by two or three plain gut sutures (interrupted) The skin is closed above and below the drain with silkworm gut, interrupted sutures. Voluminous dressings are applied, sufficiently thick to fill out the normal hollow between the head and neck.

The patient is handled with care, remembering that the brain stem no longer has its protecting cushion of fluid. The entire operation takes from fifteen to thirty minutes.

C. Difficulties of the Operation.

As to the difficulties of the operations already performed by the writer, three in number, there were none. The cisterna magna was easily and quickly reached in all cases, so easily that it demonstrated perfectly how simple the operation was. As to the emissary vein, it is not constant: it was found in one cadaver of 6, and in one patient of 3, and here bled so little that a twist of an artery clamp into the hole stopped the bleeding at once.

The division of the bone in the two children was such an easy and rapid procedure that one needed to be careful not to crowd the trephine too fast. In the adult it took about the same time as on the vertex. The separation of the dura, the removal and reintroduction of the bone cutter took no appreciable time and was not attended with a single bad symptom or drawback. As to the occipital and marginal sinuses, they need not cause one a second's thought. If in the line of the incision, tie them, otherwise not (if double).

NOTE:—In our first case a small aspirating needle was inserted through the dura into the cisterna magna and the excess of fluid withdrawn. In the other cases the structures were cautiously divided with a knife.

Regarding the danger of the escape of cerebro-spinal fluid. This danger attends this no more than any operation upon the skull. However, I think too much stress is placed upon this factor. In a fairly large experience in operating upon the skull and spine, and by perusal of the literature I am convinced that mere loss of cerebro-spinal fluid is not attended with any danger whatever. In cases of meningitis, however, where the brain is under pressure, its sudden escape might be attended with serious syncope. Yet in one of my cases there was an unintentionally large incision made into the cisterna magna and the fluid all escaped within a few seconds, yet no symptoms were observed. In the two other cases the fluid was slowly evacuated, in one through a tiny incision, in another by an aspirating needle, the stream rising in the adult to the height of six inches or more.

Our experience is not large; but in connection with that of others who have recorded their observations, it is helpful and confirmatory.

As to the danger of infection, infection is already present. The operation is undertaken to relieve the individual of its effects and possibly cure the disease. No more valid objection against operating in these cases exists than in the presence of infection in any other part of the body—even less—for often unrelieved suppuration elsewhere may spontanously open and drain itself, but, from the cranial cavity, never.

D. Advantages of the Operation.

This operation opens the cisterna magna at its largest and most favorable spot for the evacuation of any fluid it may contain. Drainage established here effectually taps all other adjacent regions within and without the brain and cord. We do not seek to establish drainage merely to provide an exit for purulent cerebro-spinal fluid, but to relieve intra-cranial pressure and restore blood to the vital centers.

Furthermore, once drainage is established, the fresh secretions of cerebro-spinal fluid are more potent in their bactericidal elements than before, and every hour gained in life leads to greater probabilities of a complete cure. (We except from this statement cases moribund at the time of operation). The operation is not complicated by the protrusion of the cerebellum into the cranial opening, as occurs in all other operations in the cerebellar fossa. "Hernia of the cerebellum" interfered with Ballance. Parkin had to "push the cerebellum out of the way" before he got any flow, beyond a few drops. Ord and Waterhouse found that the "cerebellum bulged into the trephine opening, fitting it tightly like a cork in a bottle." Hall (58), in operating upon a case after Perkins' method, says "the cerebellum protruded so forcibly that its substance was lacerated against the edges of the bone." In the operation here suggested there is no hernia cerebelli. The reasons are plain, anatomy reveals and surgery proves them.

Meningeal infection is attended with an increased amount of cerebro-spinal fluid. This causes great intra-cranial pressure. The cortical surface of the brain is crowded against the inner surface of the skull. A trephine opening made in the skull anywhere over the convexity of the brain (cerebrum or cerebellum) and the dura divided, the brain is crowded forcibly into the opening and tightly "corks it up." At the site of this operation, over the cisterna magna, and between the lobes of the cerebellum there is a normal gap or hiatus. The skull can be opened here in the presence of great intracranial pressure without a hernia cerebelli occurring or even a tendency to one appearing.

The entire central cerebello-medullary tract is under direct observation and, if required, surgical treatment. Moreover, drains can be inserted, later-

ally if necessary, for draining the lateral fossae on one or both sides. Should there be adhesions blocking up the foramen of Magendie, these can easily be broken up by merely separating the lobes of the cerebellum without disturbing the medulla. Plugging of the foramen magnum by the brain stem is impossible. The other varieties of operations suggested and performed in the sub-occipital region do not drain.

Ballance had to "trephine both sides of the middle line so as to prevent hernia of the cerebellum interfering with drainage." Parkin stated he got "free drainage up to the last," but he had to raise the cerebellum up in order to gain this. Ord and Waterhouse had a similar difficulty to contend with. Kuemmell had to trephine on both sides of the middle line to secure adequate drainage.

If a drain is inserted through a trephine opening as usually placed, the brain must be crowded back. It may be and usually is lacerated in the undertaking. If a drain is inserted it lies between the brain and skull and is pressed upon by the latter. It is soon isolated by enveloping adhesions and ceases to functionate. Drainage by the older operations is difficult and of short duration. But drainage from the cisterna magna is from cavity. It is not subjected to pressure and there is no tendency to limiting adhesions. Ordinary peritoneal drainage and its speedy isolation by adhesions resembles the former class of cranial drainage, while supra-pubic drainage of the urinary bladder by rubber tissue closely imitates the method of drainage from the cisterna magna here advocated.

As to the plan of Radmann to drain this region by an incision through the occipito-atlantal ligament, the space is very narrow, especially narrow in children, the vertebral arteries come into dangerous proximity to the field of operation, the opening is below the real seat of trouble. Relief of pressure at this point is attended with the same objection as for lumbar puncture, the possibility of "corking up the foramen magnum by the brain stem."

Furthermore, no information as to the condition within the great cistern can be obtained and there is no possibility of determining if the foramen of Magendie is closed, and no relief of such a condition, if present, could be offered.

Extra-cranial advantages are that there is a single incision with separation, merely of the muscles. There is no extensive division of muscles as in Ballance's, Parkin's, Ord and Waterhouse's or Cushing's propositions, with their subsequent resuturing—time consuming details. Nor can there be the "furious bleeding" reported by Ord and Waterhouse. Loss of any considerable quantity of blood is something to be carefully guarded against. Because it "is imperative that the blood-pressure be maintained" (Archibald), and loss of blood results in its lowering.

The operation is quickly done. Fifteen minutes in the child and, at most, thirty in the adult, is ample time in uncomplicated cases.

Further, an operation at this site will prevent future complications. Cerebral irritation and even epilepsy due to adhesions forming between the pia dura is impossible, as these two membranes are so widely separated at this point. Hydrocephalus, that frequent complication of meningitis, and probably due to the closure at the foramen of Magendie, can be prevented. As to the frequency of hydrocephalus, Lees and Barlow (59) state that in 38 out of 50 autopsies it was the hydrocephalus, and not the meningitis, that caused death.

Cushing emphasizes this condition by stating "that basal meningitis of whatever nature, provided the exudate serves to obstruct the foramen near the

fourth ventricle, is usually the immediate cause of death in the fatal cases of meningitis, and in those which recover it is a common source of cerebral disturbance, owing to an incomplete re-establishent of the normally free outlets."

Furthermore, if hydrocephalus has developed, as a sequella of "cured" meningitis, it is probably due to the closure of this outlet. By means of the operation here described, which offers greater access to this region with the minimum risk, simple technic, ease of execution, advantageous position, relief may be secured for these patients.

E. Secondary Remedial Measures.

Blood-pressure must be maintained and in no instance permitted to fall. A sudden fall in blood-pressure indicates a fatal end.

As to drugs, strychnine is our sheet anchor; adrenalin may be helpful.

Mechanical measures, bandaging the abdomen, over a small pillow, bandaging the lower extremities, lowering the head of the patient (and table if necessary).

Furnish good blood to the asphyxiated vital centers by the inhalation of oxygen.

Saline transfusions or injections during or following the operation. A Murphy drip or intermittent saline rectal enemas of a few ounces continued over the first 24 hours afford one of the best therapeutic measures. It furnishes fluid to the system, flushes the kidneys, aids the other eliminating glands and so assists in throwing off the infection.

Possibly the proper auto-vaccines or anti-toxins may be of some service in combatting the general infection. By large doses of hexamethylenamin the cerebro-spinal fluid may be rendered more anti-germicidal and thus more quickly overcome the infection.

The chief point is, however, that by early resort to surgery all these evidences of systemic poisoning may be prevented, or, if present, to some extent relieved before they have overwhelmed the patient.

F. Other Conditions in Which this Operation May Be Indicated.

I have written this paper to urge the early use of this reasonable and safe operation in meningitis. I feel, however, that it is not by any means restricted to this disease alone, but might prove a life-saver in those basal fractures extending into the posterior fossa of the skull when death is certain from pressure due to the intra-cranial hemorrhage as demonstrated by lumbar puncture and compression symptoms.

So many of these cases reach the surgeon in the terminal stage of cerebral compression and bulbar paralysis that it is realized but few may survive the operation. Is it not worth while, however, to give these otherwise fatal cases a chance? Ransohoff (60) states that in 190 cases of fracture of the base of the skull; 56 per cent. died within 12 hours; 78 per cent. within 24 hours, and 85 per cent. within 48 hours. There is thus a margin of from 12 to 24 hours in nearly 30 per cent. in some cases in which this method of decompression might be of use.

Furthermore, should infection be added, as not infrequently follows, and a basal meningitis develop, as in Kuemmell's case, would not this operation prove more advantageous than the extensive one proposed by him or similar ones suggested by other surgeons.

The indications are practically the same as in meningitis. But we do not, usually, have the opportunity of observing the cases early. Serious pressure exists, as a rule, when these cases come under observation, as shown by the

coma, fixity of the pupils, vagus pulse and irregular respirations. With such extensive bulbar paralysis operation would hold out very little chance of success. But if there was only delirium or stupor, with a rising blood-pressure, with fair pulse and respirations only stertorous and lumbar puncture gave a bloody fluid, under pressure, drainage of the posterior fossa by his route might be successful in a fair proportion of cases.

G. Instruments and Appliances.

As far as possible "stock" instruments have been selected and tried out upon the cadaver and upon the living. While we have made a few suggestions, tending to meet certain contingencies and to facilitate the operation, the entire operation may be easily performed with the ordinary instruments for cranial surgery. No chisels, gouges or hammers should be used. We have had fashioned a self-retaining retractor by a little modification of a Jansen's mastoid retractor, extending and squaring the arms and using removable tractor blades. The right and left dural ligature passers had to be made especially. There is nothing small enough that has the strength with the proper curve. The dural separators are stock instruments, slightly modified. The sharp triangular periostal scraper was found useful in separating the dura and pericranium at the foramen magnum. The rectangular bone-punch is intended for enlarging the opening in the bone, in children; in adults the ordinary bone cutters may be used for this purpose.

The hospital head-rest is constructed after the one described by H. B. Smith (61). We have aimed to make it applicable to any age and adjustable to any table.

The advantage of this head-rest consists first in securing immobility of the patient's head in the best position for work upon the region in question. Further in being attached to the table, the table may be elevated or depressed as occasion may require without disturbing the position of the patient upon it.

I have designed a portable head-rest which may be attached to any table (kitchen or operating), and is also easily adjusted to adults or children. It weighs 6 pounds while the above weighs about 15 pounds.

Chapter VI.—Records of Cases.

Case I.—A. M., Italian, 8 years of age; November 21, 1910. The child was taken sick with influenza one and a half weeks ago. Sickness began with general malaise, pains all over the body and a slight cough, no chills. Temperature 100° to 102°. Diagnosis: Broncho-pneumonia.

Ten days ago a purulent discharge appeared from both ears. There had been no complaint at any time of pain in the ears. Eight days ago the child became dizzy and four days ago had vomiting spells and then gradually became semi-comatose. Pupils did not react to light. Kernig's sign present and Babinski sign present in both legs.

At this time a petechial rash appeared over the body. Temperature, 101°; respiration, 30. Diagnosed now as tubercular meningitis. Spinal fluid was drawn and diagnosis confirmed by the Board of Health.

Dr. Kopetzky saw the child after this. He found it unconscious, pupils dilated and not reacting to light. Head retracted and rigid on body. Kernig and Babinski signs marked. He withdrew 30 ccm. of spinal fluid and sent the child to the Red Cross Hospital, November 20, 1910. Dr. Kopetzky's diagnosis: Otitic meningitis following a double mastoid.

On entering hospital, temperature, 101°; pulse, 120; respiration, 28. Blood-pressure, Janeway's cardiac sphygmomanometer, 98 to 100 mm. Hg. Operations

at midnight. Ether anesthesia by Dr. Garr, House Surgeon. Mastoid operations by Dr. Kopetzky, assisted by Dr. Imperatori.

Right side.—Outside surface markings of cortex of mastoid normal, but on removal of cortex, no mastoid cells were evident at all, the sigmoid sinus completely filled up the small mastoid protuberance. It was covered with granulations. The mastoid antrum was finally located, by pushing back the sinus wall, and the region found filled with detritus and pus.

Left side.—Nothing abnormal in structure. Pus and detritus in the process.

Drainage of the cisterna magna by Dr. Haynes, Dr. Kopetzky assisting, and Dr. Imperatori steadying the child's head. An incision was made in the middle line from the external occipital protuberance to about the third cervical spine and down to the occipital bone and the arches of the atlas and axis. Hemorrhage was very slight, four clamps controlled the bleeding. The periosteum and attached muscles were elevated from the bone and pushed toward the sides until the occipital bone was bared from the inion to the margin of the foramen magnum and an inch or slightly more in width. There was no hemorrhage.

A DeVilbiss trephine (three-eighths of an inch in diameter) was applied to the bone midway between the inion and the margin of the foramen magnum in the middle line. The skull was unusually thick in this place, but the button was turned out without difficulty and the dura exposed. This was loosened about the margin of the hole by the dural separators and a DeVilbiss bone-cutter used to make two furrows from the trephine opening to the foramen magnum. The intervening piece of bone was gradually removed by a flat rongeur. There was no bleeding. There was no appearance of any sinus, occipital or marginal. A medium aspirating needle was inserted through the bulging dura, into the cisterna magna, and enough fluid obtained for laboratory examination (about 10 ccm.).

The dura presented an injected appearance. It was carefully incised in the middle line and the cerebro-spinal fluid allowed to escape, which it did slowly but not satisfactorily. The incision was enlarged and the fluid escaped freely. It looked thin and clear.

The arachnoid presented in the wound as cotton (absorbent) looks when floating on the surface of water. It was somewhat milky in color. Nothing of the cerebellum or blood-vessels showed through it. It was severed in the middle line as deeply as I dared to go with the idea of dividing its meshes and allowing freer drainage. A rubber tissue-drain was inserted into the opening and the wound closed with interrupted plain gut sutures, No. 2. A large dressing was applied and the child returned to bed. Pulse, 144, good quality.

Comments on the operation.—The cerebro-spinal fluid did not seem to be under any considerable pressure, though several ounces flowed away. This was probably due to a lumbar puncture six hours before when 30 ccm. had been withdrawn. The meshes of the arachnoid were "fluffed" up by the fluid so as to be very conspicuous and to obscure all deeper parts. The hemorrhage was inconsiderable at all times. No sinuses were visible and there was no dural bleeding. The method of exposure gave ample room for all work. A much larger amount of bone could easily have been removed if deemed necessary.

Subsequent course.—The drainage of cerebro-spinal fluid was free. Patient roused up sufficiently to take nourishment well up to within a few hours of death. Retraction of the head disappeared the morning following the operation and the limbs became relaxed. Blood-pressure, 95 mm. Hg. The eyes reacted to light.

Patient died 34 hours after the operations of sepsis and toxemia. The heart stopped beating before paralysis of the vaso-motor center. Autopsy was not allowed.

Bacteriological.—Spinal fluid withdrawn by Dr. Kopetzky before the operations, showed a diplococcus without a capsule, probably a pneumococcus. There was, also, present in the culture, a large, unidentified bacillus. No report was made of the fluid obtained directly from the cisterna magna.

Cultures from both ears, showed diplococci. Differential count before operation.—Polys, 30; total leucocytes, 5,000; large mono and trans., 14; lympho., 56. After operation (10 hours)—polys, 30; total leucocytes, 16,500; large mono- and trans., 17; lymph, 53.

Case 2.—H. D., aged 45 years, entered Harlem Hospital, January 12; died, January 15, 1911. Patient had been sick for a week with headaches and general abdominal pain. She worked at housework up to yesterday. Last night she had severe and recurring attacks of vomiting with pain in the abdomen. She now complains of severe headaches and severe abdominal pains, especially in the upper portion of the abdomen. She was admitted as a non-urgent case, prob ably gall-stones.

Physical.—Muscular female, dusky countenance; conscious, but somewhat irrational; pupils equal, react to light and accommodation; pulse, 120, full volume, tension increased; temperature, 103°; respiration, 30. Abdomen, somewhat tender but nothing localized. Kernig and Babinski absent.

Differential.—W. B. C., 31800; l. lymph, 4; s. lymph, 9; mast cells, 2; polys, 85; January 13, the next day; W. B. C., 33,200; polys, 89; l. lymph, 8; s. lymph, 3. Widal negative.

Spinal puncture gave 15 cm. of turbid fluid under pressure. Laboratory examination:—No blood. Pus cells, large number. Micrococcus lanceolatus, positive. Kernig and Babinski both present.

On January 14, patient was more lethargic. Urine passed involuntarily. Pupils contracted and eyes deviate to left. No convulsions but general spasticity. Cervical muscles especially contracted. She was being transferred to the medical ward by my assistant when I saw the case and ordered her up for operation.

Operation.—Dr. Haynes, assisted by Dr. Boynton and the Hospital Staff. Anesthetic, ether, administered by Dr. Lumbard, hospital anesthetist, by means of the nasal tubes. Time of operation, 25 minutes. Total anesthesia, 35 minutes. Anesthetic well taken. Position on table, on face with head over the edge, as in a reversed Rose-position. Steadied in this position by one of staff.

The occipital bone was exposed by the vertical incision and bared from the inion to the margin of the foramen magnum. Not having a small trephine in working order, a larger one with a diameter of one inch was used, without any trouble, to remove a button of bone from the midline region and midway between the inion and margin of the foramen magnum, from the center of the exposed area.

The bone between the trephine hole and the margin of the foramen magnum was removed with a rongeur, opening up a space an inch wide and an inch and a half vertically.

The dura bulged from the intra-cranial pressure. It was punctured with a small knife, an opening less than an eighth of an inch being made and through this the cerebro-spinal fluid spurted to a distance of six inches or more. A care-

ful watch was kept of the heart and respirations and the flow was checked once or twice by gentle pressure of the finger, although no untoward symptoms had appeared. The wound was then drained with iodoform gauze and the muscles brought together with two or three plain gut No. 2 sutures. The skin closed with silkworm-gut.

Comments on the operation.—I did not open the dura widely in this case, inasmuch as the fluid seemed to escape so freely from the small incision. I think that this might have been done, without adding any danger to the patient or any benefit as the sequel showed.

On return to ward, patient had a general convulsion, but this was the only one she had. Within an hour the pupils reacted to light, the spasticity was less and the wound drained so freely as to soak the dressings.

Four hours later, there was a general improvement in her condition. The pupils were active and there was no deviation of eyes as there had been at a previous examination. Pulse was full and fair quality and running at 150. Respiration, 40; temperature, 102°. Morning of January 15, condition much the same. Kernig and Babinski absent. Wound draining profusely. There is a very free secretion of mucus from the nose and throat—due to nasal anesthesia. Spasticity of muscles much less, especially of the neck. Patient gradually failed and died at midnight, 32 hours after the operation. No autopsy permitted.

Case 3.—Admitted to the Red Cross Hospital, April 24, 1911. Died, April 25, 1911. Referred by Dr. L. K. Neff. Baby O'C., 11 months old; breast-fed. The infant had been sick for one week with acute meningitis in which lumbar puncture had been used several times for therapeutic purposes and by which the infecting organism was shown to be the pneumococcus. On admission, the baby had a temperature, by rectum, of 106°, pulse, 180; respiration, 40. Was in a state of coma with pupils dilated and fixed, head strongly retracted. No record of Kernig or Babinski. Blood-pressure (Dr. Imperatori) 80 mm. Hg. (Janeway's cardiac sphygmomanometer).

Blood-count: White blood cells, 8000; polys, 17; lymph, 61; large mono- and trans., 22. Operation as a last resort. It was completed within 15 minutes. Dr. Haynes, assisted by Drs. Kopetzky and Imperatori, and House Surgeon, Dr. Neff, present. Slightest amount of ether used. The operation as detailed in the previous case was performed. No difficulty in removing the bone or in opening the dura and arachnoid. The opening was made by an ordinary scalpel and was made easily and wider than intended, allowing of rapid escape of the cerebro-spinal fluid. No bad effects were noticed. Clear fluid escaped in considerable quantity. No exudate visible in cisterna magna. The operation was performed at 11:30 a. m.

On return to bed the pulse had dropped to 168, was of good quality. The Murphy drip was started. At 6 p. m., pulse, 170; respiration, 32; temperature (by axilla) 101°. At 6.33 p. m., apparently had a convulsion which lasted 3 minutes. At 8 p. m., blood-pressure, 80 mm. Hg. Four ccm. maternal serum was injected into the flank. At 11.30, temperature (axilla) 102°; pulse, 168; respiration, 36. Patient unconscious. Child died at 1 a.m., without regaining consciousness. Permission could not be obtained for a post-mortem examination.

Comments.—These three cases were in the last stages of septic meningitis. The time for operating had long passed. The operations were per-

formed as a last resort, to offer the only possible chance in a certainly fatal disease. We did not expect to save any of them. However, the demonstration of the rapidity, the ease, the freedom from any disadvantages as an operative proposition was absolute. The cisterna magna was reached, opened, drained and the wound closed within fifteen minutes in the two children and thirty minuutes in the adult. Drainage was free, profuse and continuous in all. All the symptoms were ameliorated and death came peacefully.

Operations in such extreme cases are not advocated. They accomplish nothing but an easy death and invite adverse criticism. However, as no one can as yet say absolutely when every case is beyond assistance, even in these moribund patients, I advise interference; possibly one may be saved.

These cases merely emphasize the contention of this paper that this operation must be done early. The co-operation of the family physician must be secured. He must learn that the time to operate is just as soon as the diagnosis has been confirmed by lumbar puncture. Delay invariably brings disaster. The argument that because operations in the past have accomplished almost nothing proves nothing except that there must be a change in the time, place and method of operating.

Note.—I would like to express my thanks to Dr. Emil Altman and to Dr. Lewis K. Neff for the opportunity of operating upon Cases 1 and 3.

Case 4.—Acute mastoiditis, acute purulent labyrinthitis and streptococcic meningitis. In the service of Dr. Wendell C. Phillips at the Manhattan Eye, Ear and Throat Hospital, New York.

J. H., a Greek, aged 20 years. Came under observation March 19, 1912, giving the following history: There was pain in the left ear, beginning one month ago, followed one week later by spontaneous discharge from that ear. A week before coming to the hospital paracentesis of the membrana tympani was performed. There has been no previous illness.

On admission to the hospital, March 19, he presented all the clinical signs of acute mastoiditis of the left side with infection into the soft tissues behind the mastoid process. The discharge from the ear was profuse, yellowish. The temperature at 3 p. m. was 102.4°; pulse, 100; respiration, 26. Eyes reacted to light, patient fully conscious. Tests of the labyrinth showed that it was not functionating, total deafness, absence of caloric and rotation-test reactions, spontaneous nystagmus present. At 9:30 p. m., incision into swelling below and behind mastoid did not evacuate pus. Blood pressure (Janeway) 128 mm. Hg. March 20, 9 a.m., temperature, 102°; pulse, 112; respiration, 24. Complains of intense headache. At 2 p. m., temperature 102.8°; pulse, 80; respiration, 26. Intense headache. Blood-pressure, 146 mm. Hg. (Janeway).

Lumbar puncture performed. The chemical examination of the spinal fluid showed meningitis present, although complete examination was not made (Kopetzky's test).

The fluid, while almost water-clear, showed pus cells upon cyto-diagnosis (Strong's examination), and on the next day was reported showing streptococcus present (Strong).

March 21, 3 p. m. Because of advancing signs of meningeal irritation, operation was decided upon. A radical mastoid and labyrinthine exenteration was performed by Dr. Wendell C. Phillips in the usual way and pus and detritus found, but no fistula into the labyrinthine channels was demonstrated. This was followed by drainage of the cisterna magna, by Dr. S. J. Kopetzky,

assisted by Dr. Fresner, who held the patient's head and Dr. McQuade, the House Surgeon. The occipital bone was exposed by a median incision and the parts retracted. A three-eighths-inch trephine opening made in the middle line above the foramen magnum and the wedge of bone removed by a chisel and mallet. The dura was exposed and incised. About 40 ccm. of cerebro-spinal fluid was allowed to slowly escape, its flow being controlled by pressure on the edges of the dural incision. The wound was drained by a strip of iodoform gauze and the soft parts loosely closed about the drain by four interrupted sutures. A moist dressing applied and the patient returned to the ward, no stimulation being needed. Patient was reported as comfortable, but not sleeping.

March 22, 10:45 a. m. Blood-pressure, 120-125 mm. Hg. Patient conscious, has no headache. Spontaneous nystagmus still present. (Kopetzky). 3 p. m. Blood-pressure, 130-140 (K.) Urotropin, gr. V and sodii benzoate gr. VIII. Temperature ranged from 99.2° to 101.4°; pulse, 74-90; respirations, 22-24. He slept during the following night but became restless towards morning.

March 23, 11 a. m. Blood-pressure, 148 mm. Hg. At 3 p. m., temperature, 104°; pulse, 92; respiration, 20. Blood culture taken but later report showed it to have been contaminated. Murphy drip started and continued. Patient was reported as comfortable during the night.

March 24, 1912, noon. Blood-pressure, 132 (K.) Patient had a slight nose-bleed. He was conscious. During the afternoon he became somewhat restless. His eye-grounds were examined, but no signs of pressure reported. Was restless during the night. Temperature, 103.6°; pulse, 86; respiration, 24.

March 25, 6 a. m. Temperature, 101°; pulse, 84; respiration, 24. 1:30 p. m. Patient complained of headache, but no dizziness. Nystagmus gone. Blood-pressure, 140 mm. Hg. Wound dressed by Dr. Kopetzky as drainage seemed to have stopped. One stitch was removed and the gauze drain withdrawn, followed by a spurt of clear cerebro-spinal fluid. Patient complained of intense pain in his head during this procedure. The edges of the wound were grasped and the flow controlled. The pulse dropped from 90 to 72. A rubber tissue drain was inserted.

Blood-pressure at the conclusion of the dressing was, 118-120 mm. Hg. Patient was left without any headache but still dizzy. At 2:30, an ophthalmoscopic examination showed that the fundus was normal, nerve not swollen, questionable enlargement of veins, very fine nystagmus (Dr. Oatman). He had a comfortable night.

March 26, 2:45 p. m. Blood-pressure, 125 (K.). Hypodermic injection given of 1 ccm. of the combined bacterial vaccines (450 million). At 5:30 he complained of very severe pain in his head, was vry restless. One-fourth grain morphin given. Patient rested easily until 5:30 a. m. of March 27. Again became restless and complained of pain in his head. At 11 a. m., blood-pressure, 130 (K.); pulse was soft. Blood culture gave staphylococci (probably agonal, Dr. Callison). During the night patient required one-eighth grain of morphin to control his restlessness. He died during the forenoon of March 28, perfectly conscious up to the end. No autopsy was permitted.

Comments.—In addition to this patient's mastoid and labyrinth disease he had a streptococcic meningitis. The early diagnosis of meningitis was shown by the rising blood-pressure and by the definite chemical reaction of the cerebro-spinal fluid (the absence of the carbo-hydrate-sugar) (Kopetzky), with beginning meningeal irritation. There were none of the usual signs and

symptoms of meningitis. His mind was clear, his eye-grounds were normal. His pulse and temperature only reacted to the septic infection. The patient died of septic meningitis without the usual signs of meningitis. Only at the last did he become restless and noisy. There was no stupor, slowed pulse, irregular respirations, or choked-disc invariably found accompanying the usual exitus from meningitis.

The operation distinctly relieved the patient. It did not cure because the infection was too extensive. It demonstrated, however, its value in these otherwise hopeless cases and its use is thereby justified. Some cases will be saved. It is only a question of diagnosis and early operation.

Chapter VII.—Conclusions.

Meningitis kills. The least deadly and after the best treatment 10 per cent. to 25 per cent., the other varieties, 100 per cent. of cases.

Meningitis kills by producing intra-cranial pressure, anemia of the "vital centers" and their paralysis.

Intra-cranial pressure is assisted in its effects by the constitutional results of infection—septicemia and bacteremia.

The presence of infection is early demonstrated in the cerebro-spinal fluid drawn by lumbar puncture and by the clinical effects of intra-cranial pressure, as recorded by the changes in blood pressure, fundus of the eye, pulse, respirations and state of the sensorium.

These early evidences of meningeal infection are clearly shown by: 1. Progressive increase in blood-pressure; 2. absence of carbo-hydrates from the cerebro-spinal fluid; 3. edema of the papilla (not "choked disc"); 4. a clouding or irritable sensorium.

Such findings absolutely prove infection is present. If they are present, the diagnosis is certain and the end is sure.

It takes some hours or days for death to occur.

No present therapeutic measures save more than 75 per cent. to 90 per cent. of diplococcic infections and practically none of all other varieties.

The only relief is by early, free, and continuous removal of the rapidly forming cerebro-spinal fluid, thus preventing cerebral compression, bulbar anemia and paralysis and death.

Anatomy, physiology, pathology and surgery show the cisterna magna the place best suited for such a purpose.

The operation which reaches this in the minimum of time, with the least structural disturbance, with freedom from hemorrhage, with maximum access to the region without cerebellar hernia, with provision for adequate and sustained drainage with opportunity to explore and treat adjacent parts and prevent complications and with the greatest safety to the patient is the preferable one.

The operative procedure here submitted for draining the cisterna magna fulfills these requirements more completely than any heretofore proposed or performed; therefore, it should be the operation of choice for the treatment of meningitis.

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A CASE OF EPIDURAL HEMORRHAGE COMPLICATING EPILEPSY WITH RUPTURE OF POSTERIOR BRANCH OF THE MIDDLE MENINGEAL ARTERY—RECOVERY.*

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In 1886, Jacobson, in Guy's Hospital Reports, presented a very elaborate, systematic and exhaustive study of epidural hemorrhage based upon seventy collected cases which had either come to operation or autopsy. In this report some very interesting and instructive deductions are brought out which are of no less importance at the present time than they were then.

After reading this scientific article, one is impressed by many salient points which may be briefly summarized. In all this great number of cases, no two are alike, no symptoms occurred as the outcome of trivial injuries, while others presenting few symptoms and slight injuries were the result of great violence; some presented symptoms so distinct and characteristic as to leave not the slightest doubt regarding diagnosis which was at operation verified, while other cases showed so few characteristic symptoms as to engender doubt and uncertainty relative to proper treatment.

In 26 cases where the vessel was mentioned, the main trunk of the middle meningeal was injured in only two cases, the hemorrhages occurring from some branch of the artery as a rule. The posterior branch was injured twice as frequently as the anterior. In these 70 cases, fracture occurred in 62, and in 38 cases,

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the base was involved. In 37 of the 70 cases in which the limbs were mentioned, hemiplegia was present in 19, a fact which I wish to bring out particularly, inasmuch as this aspect of epidural hemorrhage has been of late questioned (1), although the literature is full of many isolated instances of paralysis following extra-dural bleeding and is prominently mentioned by Phelps (2), Starr (3) and others.

It is for the purpose of adding further proof of the possibility of the occurrence hemiplegia resulting from extra-dural hemorrhage and because it has so many points in common with those collected by Jacobson, that the following case is chronicled, believing the many points of interest justify it.

J. Z., age 40, was admitted to Bellevue Hospital on November 25, 1910, in the service of Dr. Joseph D. Bryant to whom I am indebted for the privilege of operating upon and reporting the case. He had been subject to epilepsy for the past 17 years. Attacks occurred irregularly. Patient unconscious at these times and never knew when they were coming on. Convulsions general, with no incipient focal manifestations.

On November 24, 1910, 6.30 A. M., during one of these attacks the patient fell, striking his head on a stone walk. After the epileptic attack, the patient was assisted to a chair and after a short rest, he walked up two flights of stairs to his room. He remained in bed all day, and at 10.30 that evening took some broth which he vomited. The next morning, 24 hours after his injury, he was found unconscious in bed and sent to the hospital.

On admission the patient was in a stuporous condition, unable to talk, but opened his eyes when shouted at or shaken. He was very restless and constantly turned his head and moved his right arm and leg and made attempts to grasp those who approached him.

The left arm and leg were paralyzed, pricking and pinching these members producing no response and no attempts to move these were made, although the patient constantly moved the right arm and leg, and these responded to stimulation.

There was no facial paralysis, pupils were normal, no bleeding from ears, nose or mouth. Breathing deep, but not stertorous, 22 to the minute. Pulse 60. Blood pressure 120 on both sides.

There was a large œdematous area over the right parietal region which was very tender, the patient making attempts to move his head away when pressure was made over this. Spinal sub-arachnoid space tapped and bloody fluid obtained.

Patient was anæsthetized, and an inverted U-shaped flap turned down over the right motor area, hemorrhage being controlled by a rubber bandage around head. A linear fracture of the skull was revealed, running obliquely downward and forward over the right parietal and temporal bones into the base of skull. An opening was made over the motor area close to the fracture with a Hudson drill.

A clot was discovered as soon as a trephine opening was made and enlarged which covered the entire motor area and extended well into the base of the skull. There was no active hemorrhage as long as the clot was undisturbed, but as soon as this was removed, which nearly filled an ordinary drinking glass, an active hemorrhage was seen coming from the lower margin of the wound beneath the bone. A further removal of bone in this direction revealed the hemorrhage coming from a torn artery lying on the dura which corresponded in position and direction of course, to the posterior branch of the middle meningeal. This was controlled by ligature. The dura was intact and was not opened. The brain was markedly compressed in this region to accommodate the clot and from the gross appearance it seemed impossible that the function of the brain could ever be restored so great was the apparent indentation of the brain. The skin and fascia were closed with running catgut after inserting a rubber dam drain which was removed after 48 hours.

The patient made an uninterrupted recovery. On the following day he could move his left arm and leg when requested, but he was mentally dull; memory poor; understood questions put to him, but failed to appreciate his condition or whereabouts. Could not tell his name or address.

On the following day, however, he could tell his name; gave his correct address, and answered other questions intelligently.

He was free from epileptic attacks for three weeks after operation, at the end of which time he had one, similar to the previous attacks, while up about the ward. He had two others before leaving the hospital on December 31, 1910. Since leaving the hospital he has had many attacks at irregular intervals, he often going three weeks without one and then having two or three daily for a few days.

The points in the above case that I wish to emphasize are, that this is a typical clean-cut case of epidural hemorrhage; that paralysis of the left arm and leg were present; that the paralysis could not have been due to a hemorrhage inside the brain substance itself, since all the symptoms disappeared after the clot was removed from the dura; that the attacks of epilepsy were in no manner altered by the operation.

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THE RELIEF OF INTRACTABLE AND PERSISTENT PAIN DUE TO METASTASES PRESSING ON NERVE PLEXUSES,

*By Section of the Opposite Anterolateral Column of the Spinal Cord, Above the Entrance of the Involved Nerves**

EDWIN BEER, M.D.

Dr. W. G. Spiller has suggested (1) the operation of cutting the anterolateral column of the cord for the relief of persistent pain of organic origin and published a report of the first case illustrating this procedure, the operation being performed by Dr. E. Martin. The importance of obtaining relief for these cases is self-evident and, as I have seen a number of these patients dragging out their sufferings over months, more or less chronically poisoned with morphin, the suggestion of Dr. Spiller appealed to me. Compared wth cutting of a large number of posterior roots to effect a similar result, it seemed a much simpler and safer procedure. Moreover, if our physiology was correct, patients so operated on would lose only pain and temperature sense in the affected parts, whereas section of posterior roots (even two consecutive ones) (2) produces loss of all three sensibilities.

The patient of Drs. Spiller and Martin, a man of 47 years (1909), suffered from an inoperable growth involving the lower part of the spinal cord.

The man suffered greatly from pain in the lower limbs and required morphin every night for relief. In January, 1910, . . . the man had very slight movement at each hip and in the left knee, and no movement elsewhere in the lower limbs. These limbs were greatly wasted. Sensations of touch, pain, heat and cold were lost in the back of each thigh and in all parts of the legs below the knees, although deep pin-pricks seemed to be felt in the left calf and left foot. . . .

Jan. 19, 1911, Dr. Martin divided the anterolateral columns on both sides of the cord. January 22 there was great relief from pain in both lower limbs.

Pain was felt occasionally in the lower limbs during the three weeks following

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the operation, but the man was positive that it was less than before the operation. . . . In March, 1912, . . . his countenance expressed no pain and he never complained unless he was asked concerning his condition, when he would reply that he sometimes had pain. As he is a foreigner and not very intelligent, and his condition is pitiable, it is hard to judge whether he really has any pain from the tumor. The operation seemed to have been successful in the diminution of pain.

In the patient whose case I wish to report in this paper, the persistent right-sided pains stopped at once after section of the left anterolateral column at the level of the tenth dorsal lamina. The relief obtained was most striking, and as the patient was walking about eleven days after the operation, a more satisfactory outcome could not have been desired.

History.—The patient, Carrie F. (3), aged 43, was a kitchen helper. Family history was of no importance. In June, 1910, the patient had a hysterectomy for carcinoma of cervix. She was well till September, 1911, when she was seized with sharp and shooting pains in the right lower extremity. The present trouble had been progressive in character; beginning with severe pain in September, 1911, the pain had become worse and was referred to the right lumbosacral region as well as the right lower extremity.

First Examination.—The patient was small and thin and evidently suffering from pain. Her gait was limping, which she explained by saying pain prevented her from straightening out. She showed at time slightest hypesthesia of all three sensibilities on the right side from spine to midaxillary line from the level of the upper lumbar vertebra over the right buttock. The knee-jerks were normal, and there was no motor paralysis. The x-ray of the spine was negative. The lumbar spine, though tender (irregularly so), was not in the least fixed. The general physical examination was negative except for a tender flat mass in the right pelvis felt high up through the rectum, first detected in June, 1912.

On May 29, 1912, a test injection of stovain (4) into the spinal canal was made with complete temporary relief of all pain. This result, plus the subsequent finding of the tender mass in the pelvis, made the diagnosis clear and suggested the possibility of obtaining relief by section of posterior roots, lumbar first to sacral fifth, or by section of the opposite anterolateral tract (5).

June 29, 1912, patient continued to have severe attacks of pain in back and sacral region. Sensory examination revealed no area of anesthesia.

Second Examination (July 1, 1912).—Cranial nerves 1, 2, 3, 4, 5, 6, 7, 11, 12 appeared normal; the eighth hears watch at only 6 inches with each ear. There was no disturbance in the field of vision as grossly tested. There was slight diminution in corneal sensibility.

Upper Extremities: Good power. sensation and reflexes normal.

Lower Extremities: Negative, except for slight exaggeration of the knee-jerks. No Babinski, no Oppenheim and no clonus.

Trunk: Abdominal reflexes normal. Third and fourth lumbar spines tender but spine freely movable. No disturbance of sensibility. Sphincters intact.

Rectal Examination: Flat, very tender, hard mass in right pelvis high up.

Operation.—July 2, 1912. Dorsal laminectomy (dorsal ninth and tenth) and section of left anterolateral column were performed by Dr. E. Beer. Time of operation, twenty-five minutes. Patient stood operation very well. The cord was exposed after removal of two laminæ and incising of the dura. Nothing abnormal was noted. A strabismus hook was passed about a posterior root (left side) and the cord then rotated on its long axis by traction, thus exposing the lateral columns. Another strabismus hook was passed in front of the cord to steady the

latter while incision with a small sharp knife was made. The incision began approximately 2.5 mm. from the exit of the posterior root, the blunt side of the knife being directed toward the crossed pyramidal tract (which is very important, lest this be cut accidentally (6)). The incision was carried forward about 2.5 mm., and to the same depth. There was very slight bleeding from the section thus made. The dura was closed with silk. The muscles and fascia were closed with catgut, and skin closed over these. In the accompanying sketch the shaded tract indicates the area cut (Fig. 1).

Postoperative History.—July 3, 1912: Patient feels remarkably well and states that she has none of her old pains. There is almost complete motor paralysis of the left lower extremity, there being only slight flexion and extension in the toes, ankle and knee. The left knee-jerk is absent; the right is active.

July 4, 1912: There is slight improvement in motor paralysis. The patellar jerk is returning The distal part of the left extremity shows more motor improvement than more proximal part. There is absolute loss of pain sensibility (tested with pin) in right lower extremity.

July 5, 1912: The complete sensory status is indicated in the accompanying diagrams (Fig. 2). As will be seen, there is complete loss of pain sense over the whole right lower extremity down from the mesial aspect of the groin anteriorly and posteriorly all the way down from the middle of the buttock. In the same area

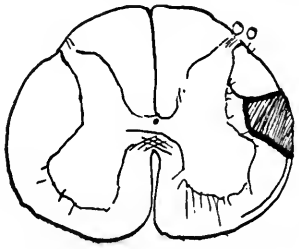

Fig. 1.—Anterolateral tract (midlumbar).

there is absolute loss of cold perception, cold being interpreted as heat. In this area heat is perceived slightly, and always as much less hot than in normal parts. Sensation of touch appears practically normal. In other words, the area affected involves all the sacral nerve fibers and all the lumbar nerve fibers up to the first lumbar, which is affected only in the lower half.

July 8, 1912: Sense of position in all joints is very accurate. Perception of pressure, as in squeezing the muscles of the right lower extremity, is slightly impaired, pressure being less distinctly perceived than in the left lower extremity. The knee-jerk in the left leg is much stronger. Motion is much improved.

July 10, 1912: Dressing. Sutures removed. Good union. Motion in left leg improving steadily. Sense of position in both legs normal. No further change in loss of pain sense in right lower extremity. Palpation of pelvic mass, which caused severe pain before operation, not painful now.

July 14, 1912: Examination shows loss of pain sense as on diagrams. Disturbance in thermal sense as before, except that area has contracted about 1 or 1.5 cm. downward. Pressure sense less acute than in normal extremity. Sense of position of joints is normal in both lower extremities. Both knee-jerks are active. There are no trophic disturbances, no bladder or rectal symptoms and no motor weakness in right lower extremity. Patient is able to walk; motor power in left lower extremity has returned almost completely. All pain in right lower extremity is gone and no vestige of it felt since operation.

July 16, 1912: More careful study of quantitative changes in touch sensibility in right lower extremity suggests a very slight diminution in perception of touch as compared with the other normal extremity.

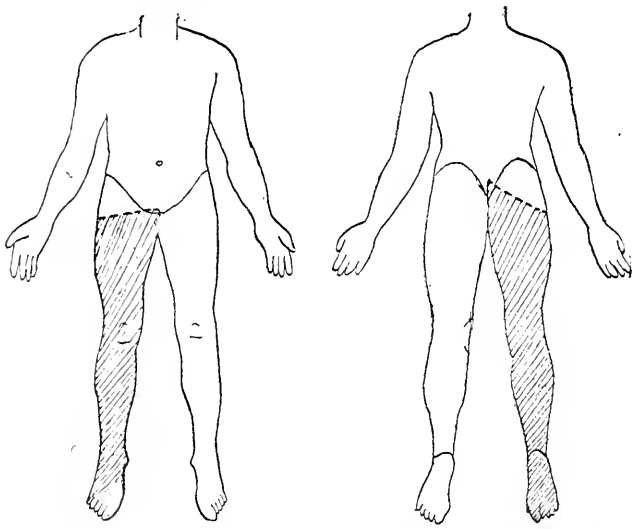

Fig. 2.—Diagrams of extent of pain anesthesia.

July 18, 1912: During the past four days there has developed an edema of the right lower extremity, probably due to pressure of the growth on pelvic vessels. This makes locomotion more difficult. Deep pressure over the tibia may produce some pain. While the patient is lying in bed the heel-to-knee test of the lower extremity brings out an ataxia which, however, is almost entirely absent when the same test is made sitting in a chair. How much of this is due to a real ataxia and how much to the marked edema and consequent heaviness of the extremity cannot be decided at the time. There is no disturbance of this kind in left lower extremity.

July 21, 1912: Repeated attempts to produce pain (deep) by firmest pressure on tibia were unsuccessful (pressure producing subcutaneous bleeding). The ataxia in the right lower extremity has disappeared with the disappearance of most of the edema under firm bandaging. Patient walks about ward; occasionally complains of pains on the left side; never has the least pain on the right side or in the right lower extremity.

July 26, 1912: Condition continues the same. Patient fell in trying to step over window-sill and bruised herself, causing some pain in her back.

Note by Dr. Richmond.

Aug. 1, 1912: Patient complains of pains in the back and says that she cannot sleep. It is relieved by ½ grain of codein by mouth. Walks about the ward, but appears weak, especially in the right leg.

Aug. 3, 1912: Patient complains of general weakness and pains, and has pain in the back when lying flat. On percussion of lumbar spine tenderness is elicited. Patient is emotional and despondent. There is increased frequency of urination and burning in the urethra.

Aug. 6, 1912: Condition about the same. Patient is very restless at night, but sleeps after administration of codein or salt water. Examination of right leg shows absence of sense of pain, just as after the operation.

Aug. 12, 1912: For the past days the patient has been very comfortable, sleeping well and having no pains. She is up every day, either walking with a crutch or resting in chairs.

Aug. 20, 1912: For the past eight days the patient has been for most part very comfortable, but is emaciating steadily, while the tumor is growing.

Sept. 5, 1912: Weakness and emaciation are progressing rapidly. Patient is in bed most of the time and has pain in her back. Marked edema of the right leg and of the labia has developed. The recurrence in the pelvis is much larger, extending up to the umbilicus, and is not tender. Sensations in the right lower extremity are unchanged. The pain in the back is constant with slight remissions.

Sept. 15, 1912: The pain is less. Patient is in bed all the time, with steady progression of all pressure symptoms on vessels, bladder, etc.

Sept. 22, 1912: Patient appears somewhat stronger to-day, and during past week. Pains in the back recurred to-day.

Sept. 29, 1912: Patient has been up in a chair for the past six days, and though the pains in the back have again become less, there is distinct progression of the cachexia.

Oct. 1, 1912: Condition is unchanged. Patient sits up in chair most of the time.

Oct. 24, 1912: Pain in the back has returned; there is none in the region of analgesia. Patient cannot walk without assistance and is getting weaker.

Nov. 15, 1912: Patient gradually has become more and more weak, and several days before her death she passed into a comatose state.

Conclusions.

1. Surgically considered, the operation of cutting the anterolateral column without doing serious damage to the rest of the spinal cord is perfectly feasible and not difficult.

2. Therapeutically considered, the almost complete freedom from pain (7) produced by this surgical intervention met the indications presented in the case here reported, and in many other sufferers a similar intervention, I feel sure, will give similar relief.

3. Physiologically considered, section (complete?) of the anterolateral column produces (a) loss of pain sense on the opposite side of the body; (b) a disturbance in thermal sense which suggests that the fibers for heat and cold are disassociated in the cord; (c) slight disturbance of deep pressure sense and slightest disturbance in touch sense, both being impaired without producing any disturbance of sense of position.

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1. Spiller, William G., and Martin, E.: The Journal A. M. A., May 18, 1912, p. 1489.
2. Seen in one case of posterior root section in 1910, in which the fifth lumbar and first sacral were cut.
3. For the transfer of this patient to the surgical service I wish to thank Dr. C. Strong.
4. The importance of such a test cannot be overestimated. If stovain or novocain, etc., produce relief, the operation promises relief. If they fail the operation is contra-indicated, for self-evident reasons.
5. For reasons mentioned above, the latter method was chosen.
6. Compare Dr. Martin's technic with double-edged knife.
7. All pains in extremity had ceased. Whether a higher section would have abolished the pains that developed at a higher level along the spine, further experience must demonstrate.

A CLINICAL STUDY OF RENAL FUNCTION BY MEANS OF PHENOLSULPHONEPHTHALEIN*

E. L. Keyes, Jr., M.D., and A. R. Stevens, M.D.

In May, 1910, Rowntree and Geraghty offered before the American Association of Genito-Urinary Surgeons an exposition of the advantages of phenolsulphonephthalein as a test for the functional activity of the kidneys, and published this two months later (1). In this communication they tabulated the results obtained with these tests upon 130 patients, upon many of whom other functional tests were employed by way of comparison. As a result of this comparison they maintained that the phenolsulphonephthalein test has many advantages over the tests previously employed.

A few weeks after this report was made, Dr. Stevens began a series of observations upon the efficiency of phenolsulphonephthalein as a renal function test, and in October Dr. Keyes joined him. We have applied the test 154 times (coupled with ureteral catheterization in 40 instances) to 100 cases.

We propose to record our experiences with the technical difficulties, with a few variations in methods, and our observations on patients with supposed normal kidneys and on several groups of pathological conditions.

Technique.

As shown by Abel and Rowntree, this drug is excreted almost exclusively by the kidneys. It is readily identified in urine, even in bloody urine, by its brilliant scarlet color in alkaline solution— the alkalinity being obtained by the addition of a few drops of a 25 per cent. sodium hydroxide solution.

In all our observations, 6 mg. of phenolsulphonephthalein (1 c.c. of solution) has been used uniformly. It is injected subcutaneously, or better intramuscularly (exceptions in our work are referred to later), the time noted, and the first appearance of a pink tint attentively watched for as urine drips from a catheter previously inserted in the bladder into a vessel containing a little alkali. The catheter is withdrawn and the collection of specimens begins with this first appearance of color. We have as far as possible collected urines for two full hours at intervals varying from 1-4 hour to one hour. At the end of each interval of time, catheterization may be resorted to, or the patient may be allowed to void, provided there be no residual urine. It is better, though not necessary, to give 200 to 400 c.c. water before the test, to ensure the secretion of larger quantities of urine, merely to minimize the error due to the loss of small amounts during catheterization and manipulation. Especially is this advisable before ureteral catheterization (or during the first half hour, in order to combine the experimental polyuria test), as this procedure tends to overcome the functional anuria so often induced by cystoscopy. We have obtained large percentage of phenolsulphonephthalein from small amounts of urine and confirm the belief of Rowntree and Geraghty that additional water does not increase the output of phenolsulphonephthalein.

The percentage of phenolsulphonephthalein injected hypodermically which is contained in the several urine specimens is determined colorimetrically by means of the Duboscq Colorimeter. A trial by one of us of another and less expensive instrument (Schreiner's) convinced him of the unreliability of the latter for this use. A standard for comparison is obtained by adding 3 mg. of phenolsulphonephthalein to a liter of distilled water, made thoroughly alkaline with caustic soda. We agree with Rowntree and Geraghty that this strength of solution affords the

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most convenient one, and that in general the most satisfactory results are obtained when the indicator corresponding to the plunger lowered into the "standard" reads 10. However, often, especially in dealing with relatively small amounts of phenolsulphonephthalein or relatively large proportions of other coloring matter, we have changed the standard to 5 and feel confident after many comparative tests that not seldom this is the better procedure. A solution of 1½ mg. to the litre has been tried, but this adds an unnecessary complication and reducing the standard from 10 to 5 gives practically the same results.

The urines to be tested are each diluted with water to varying amounts up to 1000 c.c., depending on the depth of color, NaOH is added till there is no further deepening of color, and lastly the mixture is filtered. The degree of dilution best suited to an individual specimen is a matter of experience; the effort should be made to compare columns of fluids of approximately the same height. The color of the urine containing phenolsulphonephthalein is rarely of just the same hue as the standard (with water as the diluent). The pigments of the urine impart a reddish tinge, more marked of course with deep amber urine and with small percentages of phenolsulphonephthalein. Blood gives a brownish red tint. A test solution made of urine instead of water could be used to obtain a better color for comparison but would have to be renewed daily (whereas the water-standards remain constant a long time), and in practice this procedure is unnecessary. Chart I indicates the degree of accuracy of the routine method. Phenolsulphonephthalein was added to eight flasks each containing 25 c.c. of clear deep amber urine, in proper amounts to make 50% (of 6 mg.), 25%, etc., to ½%. These various mixtures were then diluted and treated precisely as in the usual estimations. The readings made immediately and two days later are indicated on the Chart (I), also the faded condition of each mixture after standing four weeks by a window in the sunlight a few hours each day. In 25 c.c. of highly colored urine, two to three per cent. is not easy to read correctly, and smaller percentages appear as "traces." A small drop of blood added to 15 c.c. of the 50% and 25% mixtures on the second day altered the readings but little practically—50 instead of 53.2 and 18% instead of 21.4%. That the percentages of phenolsulphonephthalein in urine do sometimes change on standing is indicated on Chart I and needs emphasis. Urines (infected) from case 86 showed 26.3% and 17.9% of phthalein the first day after collection, 22.7% and 10.0% the fourth day, and 16.7% and 8.9% the seventh day. Hence readings should be made within 24 hours after collection of specimens, if possible, surely within 48 hours.

Rowntree and Geraghty recommend the 2 c.c. Ricord syringe. It is the most satisfactory in every way except for the great care necessary to avoid breakage during sterilization. We compared seven syringes representing five makes with a standard 1 c.c. glass pipette such as is used in chemical laboratories. The "Ricord" cubic centimeter was .96 of that measured by the pipette, and the other syringes recorded .70, .80, .80, .88, .90, .90 of a cubic centimeter. These absolute variations are of no consequence if one will adopt these suggestions. Use only a tight syringe with a smoothly gliding plunger. Use the same syringe for all cases. Use this syringe to measure the phenolsulphonephthalein in making up the standard solution. Any error in absolute measurement of the syringe is carried along in the preparation of the standard, and the final percentage readings will be correct.

To avoid inaccuracy due to evaporation during sterilization of the phenolsulphonephthalein solution, it has been our practice to sterilize a convenient portion and make a standard from this. A new standard was thus made after each such sterilization. As a matter of fact, these different solutions were frequently compared and always but once read within one to two per cent. of one another. In one instance, however, the standard seemed to have faded 5 to 10% in two months.

As to the site of injection, we have used many regions of the body, but avoided any oedematous spot. Whether the results depend on the site of injection our data will hardly answer. In eight cases giving a practically normal output, in whom the deltoid muscle was used, the color appeared on the average in 7 minutes. In seven similar instances, in whom injections were in the thigh, the color appeared in 8½ minutes. The outputs for the first and second hours in the two groups were practically the same.

Normal Cases.

Rowntree and Geraghty found as a result of 27 tests upon 21 patients with apparently normal kidneys that the drug appeared in from 5 to 12 minutes, that, as measured by the Dubuscq Colorimeter, from 41.6% to 66.6% was excreted within the first hour thereafter, from 11.9% to 26.5% in the second hour; a total for the two hours of from 61% to 85.8%.

Our eleven observations (with intramuscular administration) upon eight cases with probably normal kidneys gave similar but lower figures. The onset of excretion varied from 5½ to 12 minutes; excretion in the first hour, 35.7% to 57.9%; during the second hour, 5% to 16.7%. total for the two hours, 49.4% to 71.2%. (See Chart II). After the second hour the percentages are always very small, often only traces, at most (in six observations) only 4% during the third hour. Thereafter one finds traces for variable periods (from 5 to 8 hours) in normal cases, though when renal disease restricts the earlier output, the later amounts are relatively higher.

Thus one needs measure only the output during the first two hours after the appearance of color in the urine (made alkaline). Whether the average normal excretion for the first hour, second hour and total two hours, shall be set as high as 52.3%, 19.0% and 71.3%, as given in the original series, or 47.0%, 10.2%, and 57.2%, as in ours, the future must decide. We can say that our observations upon abnormal kidneys and kidneys suspected of disease confirm our opinion that 40% for the first hour and 50% for the first two hours is in practical work a sufficient output for two normal kidneys. It should be noted, however (Chart II) that our group of normal kidney cases had some minor ailment or had recently recovered from such. These pathological conditions may have diminshed the excretory functional capability of the kidney tissue, which would account for our lower figures.

It seems that probably this test is of great delicacy and surely does show variations in the same individual which cannot be accounted for clinically. These variations in figures, when these figures are relatively high, we believe are of no practical importance, bearing in mind the wonderful reserve power of the kidneys. The functional capacity of normal kidneys is probably varying from time to time, with physiological processes, and the limits within normal, of these variations, we do not know.

For the purpose of estimating the time of maximum output of phenolsulphonephthalein after intramuscular injection, there are arranged in table III four cases with practically normal kidneys (output in each over 60% in two hours), in whom the collections were made at half hour intervals. The kidneys excreted on an average in the first half hour after the appearance of color in the urine, 32.3%; in second half hour, 18.2%; in third half hour, 9.8%; in fourth half hour, 4.8%. That is, over 50% of all the drug excreted in two hours was recovered in the first half hour.

Phenolsulphonephthalein was administered intravenously to four patients (cases 8, 10, 21, 104) in all of whom the intramuscular method was also employed. In every instance the drug appeared in the urine at least two minutes earlier when given by the intravenous route; and a larger percentage of

the total amount recovered, appeared in the first hour. The shortest time of appearance was 3 minutes (case 104—9 days after nephrectomy for tuberculosis). In this instance, the specimens obtained at quarter-hour intervals showed in the first hour, 18.6%, 31.1%, 8.0%, 3.8%; specimens at half hour intervals during the second hour gave 3% and "trace." 77% of all the drug recovered in two hours appeared in the first half hour. Obviously, if one employed intravenous injections, briefer intervals than one hour would be necessary.

We have found a very grave source of error in studying the total kidney function from specimens obtained by ureteral catheterization—and one seemingly overlooked by Rowntree and Geraghty. This is retention of the drug as a result of the functional disturbance incident to ureteral catheterization, which however, does not seem to vitiate the accuracy of the ureter catheter readings as compared with each other. That this functional disturbance may upset all percentage calculations, whether of nitrogen, urea, freezing point, or phloridzin, has long ago been noted by Kapsammer and others. The error is readily disclosed by a control observation taken the day before or the day after cystoscopy.

Singularly enough, in the employment of various tests of renal function, with none of them have we found pre- or post-cystoscopic control more necessary than with phenolsulphonephthalein. We would emphasize that the retention of the drug under these circumstances apparently does not alter the value of the test in a differential comparison of the two kidneys, but that the repetition without cystoscopy is often found necessary to obtain correct notions of the absolute renal function.

For example, a case of polycystic kidneys (No. 62) showed on February 2, 1911, 7% from the right kidney and 3% from the left one in one hour, and only a trace from the bladder at the end of the second hour. On the following day the test was repeated without cystoscopy. It then showed 38.5% in the first hour, 17.9% in the second, 56.4% in all. Two days thereafter ether was administered and the right kidney removed. The patient's convalescence was entirely uneventful.

Another case suspected of renal tuberculosis, when cystoscoped under spinal anesthesia showed only traces of phenolsulphonephthalein in the various specimens examined. Indeed so faint was the color in the first half hour that it was difficult to determine the precise time of onset of the drug. Yet the next day the color appeared brilliantly in 8 minutes, and 44.6% was excreted in the first half hour, 6.6% in the second, 51% in all.

Another case, this time a man with very mild bilateral bacillus coli pyelitis, gave by ureter catheter in one hour 4.3% from the right kidney, 6.25% from the left, and a trace from the bladder; total 11% in one hour. Four days later, without cystoscopy, he excreted 40.4% in the first hour, 8.6% in the second, 49.0% in all.

In one instance, however, one of us, not suspecting the possibility of this error, catheterized the ureters of a patient with polycystic kidneys, obtained but traces of color in two hours, and prophesied that the patient would soon be dead. She thereupon engaged as a scrubwoman in Bellevue Hospital, worked there for two months, and then left in a huff, exhibiting every sign of mental and physical vigor.

Since such a marked inhibition of phenolsulphonephthalein excretion may result from ureteral catheterization one naturally infers that the same phenomenon may occur without cystoscopy, and may render the ordinary, non-cystoscopic readings inaccurate. That such a disturbance does not often

occur is obvious, but that it may occur seems to us probable. But a non-cystoscopic reading requires but little manipulation to terrify, or physically to disturb the patient. The hypodermic injection is as painless as may be, and as a rule (if there is no residual urine in the bladder) the only other instrumentation required is the passage of a catheter to determine the moment when the color appears in the urine. Yet we have reason to believe that even this (whether by urethro-renal reflex, or by psychic inhibition, we do not know, and for our present purpose it does not matter) may exceptionally cause sufficient inhibition to vitiate the test. Such inhibition must be rare, yet the possibility should always be borne in mind, if the phenolsulphonephthalein test gives results contradictory to those derived from other tests, or from clinical observations. Under such circumstances it should be repeated.

The possibility of this inhibition is moreover only one of many reasons why it might be practicable to omit all notice of the delay in appearance of the drug in estimating percentages, and to count the hours from the time of injection instead of from the time when the drug appears in the urine.

It is true that marked inhibition of phenolsulphonephthalein excretion by renal disease is often associated with marked delay in its excretion, yet this delay is totally unreliable, and has been a negligible factor in most of our cases. Rowntree and Geraghty themselves concur in this.

Medical Cases.

We have classed 19 of our patients as medical cases (See Chart IV). The first of this group (case 29) was a man 75 years old, who at the time of the test presented a typical picture of broken cardiac compensation in extremis—large heart, very marked oedema of lower extremities, Cheyne-Stokes respirations, small, weak pulse, and practically unconsciousness. The output, 35.2% in the first hour, seemed amazingly large. This experience stimulated work on medical patients of the cardio-vasculo-renal group, and gave rise to the hope that the use of phenolsulphonephthalein might prove a valuable factor in differentiating the primary cardiac from primary renal cases. Such a study to be of value should be substantiated by careful necropsy examinations. We were able to secure but one such (Case 85). The phenolsulphonephthalein test was done the day before death. Color appeared in the urine in 11 minutes; 14.5% of the 6 mg. injected was recovered in the first hour and 8.0% in the second hour. Clinically, the case seemed primarily cardiac, with dyspnoea and marked oedema of lower extremities and Cheyne-Stokes respirations. The urine showed a trace of albumin, and a few granular casts. Blood pressure was 155 mm. Hg. three days before death. Post-mortem there was double hydrothorax; the left ventricle was markedly hypertrophied and somewhat dilated; the right ventricle much dilated; the mitral valves thickened; the aortic cusps fused for 4 mm. on each side and thickened. Both kidneys were small, cortex of moderate thickness, markings indistinct, capsule stripped, leaving slightly granular surface. Microscopically there was general chronic passive congestion, and in different parts of each kidney were to be found sometimes a normal appearance, sometimes acute inflammation, again chronic inflammation, the fibrous tissue crowding the tubules. Some glomeruli were normal, others had undergone complete hyaline degeneration. On the whole, there seemed a fair amount of functioning kidney parenchyma. The case is not a very striking one, but the findings seem to us to tally with the prediction of the test. The amount of the drug recovered in two hours (20.5%) was rather low, but not indicating an immediate fatal issue from renal deficiency.

In the absence of pathological proof, we shall not discuss the Nephritis cases, clinically so diagnosed. Diagnosis of this group of diseases and the findings after death are too often at variance. The data are recorded in table VI. Two patients, pregnant 4 and 5 months, respectively, both having albumen and casts in the urine, are of interest because of the relatively high excretion, in each case over 50% of the drug in two hours. Three cases of polycystic kidneys (Nos. 56 and 62 of table IV and 61 of table IX) were tested with phenolsulphonephthalein during ureteral catherization and all gave exceedingly small amounts; in but one of these was a second test made, without cystoscopy, and the output in two hours was 56.4% (contrasted with 10+% the day before during ureteral catherization). Our inability to repeat the test on the other cases leaves us in the dark concerning the absolute functional renal capacity of these patients.

The most striking fact in this group of medical cases is the uniformly large amount of phenolsulphonephthalein recovered from cardiac patients with broken compensation, and critically ill. All had albumen in the urine and usually casts were also recorded.

Miscellaneous Cases.

Table V gives, in tabulated form, data on a miscellaneous group of cases, most of them with lesions of the lower urinary or the genital system. This work is confirmatory of our general conclusions. The principal facts are stated in the table. No further elucidation seems necessary in this place.

Results in Prostatic Hypertrophy and Carcinoma.

Let us now consider the results obtained by the phenolsulphonephthalein test in cases of prostatic hypertrophy and carcinoma.

Drs. Rowntree and Geraghty studied 53 such cases, about half of them operated upon after the test. We have made 33 tests upon 17 patients, 9 of them operative.

Rowntree and Geraghty observe that "taken in conjunction with the clinical conditions, it (this test) is of more value than the study of urine output, total solids, total nitrogen, and urea estimations" (p. 627).—"A marked decrease in the amount eliminated almost invariably means severe derangement of renal function" (p. 627).

"When the time of appearance is delayed beyond twenty-five minutes and the output of the drug is below 20 per cent. for the first hour, operation is postponed regardless of the patient's clinical condition. If, under routine treatment, the output remains low but constant, the renal function is probably in a stable condition, and the operation may be undertaken, care being taken to select an anesthetic which will not further depress the renal function. In one instance a successful operation was performed with an output of 8 per cent. for the first hour, but this output had remained constant for a period of five weeks. The low output here was ascribed to chronic interstitial changes in the kidney, and nitrous oxide was accordingly employed."

"When the residual urine is large and the patient has been leading a catheter life, even if the output at a single determination is large, operation is deferred in order to determine whether the functional activity is stable, for it has long been recognized that following the relief of retention the function of the kidney is extremely variable. Repeated determinations should be made, and, except when unavoidable, operations should not be performed when the tests indicate a decreasing function. There have been two such cases in our series in both of which operation was followed by death from acute suppression."

"Again, when only a trace of dye is excreted, operation should not be attempted, as grave renal changes exist. Two cases excreting only a trace died of uraemia within a short period. In neither case was any operation performed, though clinically at the time of the first test no evidence of uraemia was detected." (pp. 657-8).

In our work (see Table VI) the time of appearance of color has not been a factor of much assistance. In general, the patients who give a lower output of phenolsulphonephthalein have a longer lapse of time between the injection and the appearance of the drug in the urine. The times of delay in three striking instances of low output (Nos. 22, 24, 94) varied betwen 18 and 50 minutes. We ventured the opinion above, that it might be a satisfactory working scheme to begin the time of collection of urines from the time of hypodermic injection, neglecting this interval of "delay," inasmuch as this figure alone is not to be depended upon as a guide. In such an event, obviously a new series of normal cases would have to be studied. However, this interval is usually easily ascertained, affords one more (though minor) point of interest, and includes the time of absorption (from injection to the presentation of the drug to the kidney cells), which is probably a very variable factor in ill patients, and one we should like to differentiate from renal excretory capability. The very long periods of delay (1 to 1½ hours) in five tests on case 95, with relatively good percentages of the drug recovered in each of the two following hours is suggestive of an occasional possible source of error if the simpler procedure were adopted.

Concerning operation in the face of low excretion of phenolsulphonephthalein, cases 24 and 94 with but traces (2-3%?) and less than 5% in two hours, respectively, died of typical uraemia on the 7th and 5th days after prostatectomies, the exit of case 94 being hastened by a terminal pneumonia. No. 24 was a feeble old man of about 65 years with benign prostatic hypertrophy, a poor operative risk clinically but with no uraemic signs prior to operation. He stood preliminary suprapubic drainage but succumbed to subsequent perineal prostatectomy under spinal anaesthesia. No. 94 was an older patient (said to be 80 years) with a benign hypertrophy but in better clinical condition. We advised against immediate prostatectomy, basing advice on this test, but the operator felt justified in going ahead, and removed a calculus and the prostate suprapubically. The patient died on the 5th day, uraemic.

In contrast to these two cases, No. 22 is most instructive. This man of 55 years, with pasty pale color, had had much vomiting and chills during the winter of 1910, and had lost considerable weight. His urine contained nearly 5% of albumin (by volume). On October 29 and November 6, phenolsulphonephthalein was injected, and color did not appear under 45 minutes. In the subsequent two hours on the latter date, but 5.5% of drug was recovered. Suprapubic drainage under cocaine was performed November 8th, and the drug output had risen to 8.+% on November 11th, Under spinal anaesthesia, perineal prostatectomy was performed on November 15th. Convalescence was quite satisfactory and the excretion of phenolsulphonephthalein for two hours rose to 17.0% on December 9th. The preliminary bladder drainage and use of spinal anaesthesia are regarded as extremely helpful features in the conduct of this case, who we believe would probably have died had immediate prostatectomy under general anaesthesia been done.

The other operative cases gave good outputs and did well subsequently. In case 91, this function test was a distinct guide. The operator had de-

clined to interfere, basing his judgment on clinical appearance and examination, but did perform perineal prostatectomy under general anaesthesia on the strength of our report. The outcome thoroughly justified the advice.

A substantial increase in excretion of phenolsulphonephthalein after prostatectomy is noted in cases 22, 25, and 26, and after suprapubic drainage in No. 17 showing improvement in renal excretion for this drug at least. Case 8 (benign hypertrophy) showed no such improvement in renal function after prostatectomy. This man, however, had been dependent upon and had used a catheter regularly for 20 years. Moreover, as proved by ureteral catheterization 8 weeks after operation, both kidneys were free from infection. In such an instance, one would hardly expect removal of the obstruction in the lower tract to materially benefit the renal excretory function, at least after so short a time.

Urea percentage and total urea of a specimen of urine collected during a brief interval of time afford no estimate of the combined function of a patient's kidneys. If it were worth while, abundant proof of this could be extracted from these records. But more dependence is usually placed upon the urea percentage and total urea of a 24 hour collection. We have arranged nine cases with these data in table VII, and make the reference here inasmuch as two prostatic patients afford our most conclusive basis for comparison of this method of estimating renal function with the phenolsulphonephthalein test, which comparison is in favor of the latter. One case (clinically in very poor condition) gave 13.1 grams of urea one day and 38.5 grams a few days later, while our color test gave uniformly low figures on corresponding days, thereby agreeing with the clinical facts. The other patient, who died after operation of definite uraemia, gave before operation only traces of phenolsulphonephthalein but 16.6 grams of urea in 24 hours (within normal limits!).

Our experience with hypertrophy and carcinoma of the prostate leads us to subscribe most heartily to the following principles. We find that—

(1) The phenolsulphonephthalein test does indeed indicate renal deficiency more accurately than any other urinary test; and that—

(2) Operation is contraindicated when only traces of the drug appear in two hours after injection.

But we disagree with Rowntree and Geraghty upon the following points:

(a) We do not recognize a diminshing phenolsulphonephthalein output as an absolute contraindication to operation (note case 8, table VI).

(b) We do not pretend to know how low the phenolsulphonephthalein output may be and yet the patient survive prostatectomy. In one case we operated successfully upon a patient under spinal anaesthesia who excreted only 2.5% in the first hour (after 50 minutes delay) and 5.5% in the second hour. Moreover—

(c) We feel that apart from hexamethylenamin and water before operation (both of which may be overdone), the patient's greatest safeguard, in desperate cases, lies in preliminary drainage by suprapubic or perineal section, followed, after an appropriate interval, by prostatectomy under spinal anaesthesia.

The Test in Surgical Renal Disease.

The conclusions reached by Rowntree and Geraghty from a study of 17 cases of renal infection, (of which 6 came to operation) by urether catheterization and other tests are as follows (p. 659):

"It has been demonstrated that the time of appearance and the percentage output is practically the same for the two healthy kidneys. When only one kidney is diseased, the time of the appearance of the drug is delayed on the diseased side and the amount excreted is not only relatively but absolutely decreased. The amount of delay in the time of appearance is comparatively of little value. Reliance is only to be placed upon the quantity excreted during a period of at least one hour. It is possible by using large doses and extending the observations for a period of two hours, each side being collected separately, to demonstrate in some degree the reserve functional ability of each kidney.

"Although in the majority of these cases of unilateral disease the combined output is equal to that of two normal kidneys, the greater part of the excretion is shown to be performed by the healthy kidney. In proportion to the decrease in function on the diseased side, approximately there is a proportionate increase in the function on the healthy side. In such cases following nephrectomy the remaining kidney eliminates an amount of drug which is normally excreted by two healthy kidneys. In all cases studied the output from the remaining kidney has been greater than the combined output from the two kidneys prior to operation.

"In one case of pyelitis no disturbance of function was indicated."

We have studied 33 such cases, five of whom had previously been nephrectomized, and five others upon whom, for various reasons, the phenolsulphonethalein test was not employed in conjunction with the ureter catheter, leaving 23 upon whom the combined test was applied. Ten patients with renal infection (tables VIII and IX), and one with cystic kidneys not infected (case 62, table IV) submitted to nephrectomy after the test. We have based our deductions chiefly upon these cases.

The technic of the test with ureteral catheterization is not complicated but the interpretation of results requires close attention. One should use as large ureteral catheters as convenient; in our experience the flute-tipped ones drain well and allow the least extra-catheter flow. With the best of catheters, this leakage may occur at any time. Accordingly, the results are of greater value if the bladder contents (if any) be obtained at the end of each period of collection. If but one ureter be catheterized, the bladder will contain the secretion from the other side plus extra catheter flow. A dilated renal pelvis (with residual urine) may greatly vitiate one's interpretation of the test. We do not for one moment neglect the usual chemical and microscopic examinations and urea estimations. The latter figures compared with the output of phenolsulphonephthalein in individual specimens help mightily to clear the skies in some instances.

We shall consider some of the more instructive operative cases briefly. No. 61 (table IX) with cystic kidneys, one infected, presented but traces of color in two hours after an injection of the usual 6 mg. of phenolsulphonephthalein, yet survived nephrectomy admirably. This case does not discredit the test, however, for the following reason: The patient was a nervous, apprehensive, mistrusting individual and unfortunately the only test made was applied in conjunction with ureteral catheterization, the marked inhibitory effect of which procedure has been already discussed. Cases 42 (table VIII) and 67 (table IX) interested us particularly because of the falling output of phenolsulphonephthalein before operation—from 45.2% for two hours in December, 1910, to 15+% in April, 1911, in the former (R. renal tuberculosis); from 21.8% to 17.4% in the latter (L. renal calculus). The amount of phthalein excreted just before operation in each case was rather small, yet not small enough for this factor alone to

be regarded a contraindication to operation. Both stood nephrectomy well and subsequent tests showed gains, a very striking increase in No. 42. The renal calculus patient had pulmonary tuberculosis, which unhappily became acute after a few days, and was regarded as the chief cause of death on the sixteenth day following operation.

The increase of phenolsulphonephthalein output after nephrectomy is a striking confirmation of the clinical observation that one good kidney alone (after operation) does better work than a normal kidney and a diseased one combined. Case 42, just cited, is illustrative of this fact, and from No. 75 (table VIII) we recovered 29.4% of the drug in two hours before operation, and 38.1% in two hours just three weeks after.

Two other deaths remain to be recorded. Case 27 was one of severe infection of a horseshoe kidney of seven weeks' duration who died about eight days after operation, which could be little but an exploratory one. The output of phenolsulphonephthalein was 25.3% in two hours, four days before operation. That the individual could survive the immediate effects of operation was about all one could expect any function test to indicate in such an unfortunate condition. Case 45 tells another story. The man, aged about 50 years, had had clinically R. pyonephrosis for 4 years. Before operation his condition seemed perfectly good. On January 3rd the phenolsulphonephthalein excretion was delayed 13 minutes, was 38.4% for the first hour and 16.6% for the second; on January 13th he voided 1600 cc. of urine with 1% urea in 24 hours. Cystoscopy had shown the R. kidney to be the source of the pus, and R. nephrectomy was accordingly done on January 7th. In 50 hours after operation the patient voided but 20 ounces of urine; the bowels moved freely; there was repeated vomiting. The pulse became irregular and rapid at times. He had "air hunger," and without showing any nervous symptoms or delirium died 53 hours after operation. The tongue was moist an hour before death. The remaining kidney was removed post-mortem and examined by Dr. Symmers, who reported as follows:

"Specimen consists of a kidney 10 c.m. in length. Capsule is thin and surface is smooth, except for a few retained fetal lobulations. The organ is diffusely bluish-red in color, and on section cuts readily. Cut surface is smooth, deep-bluish-red in color and drips blood on pressure. The consistence is that of a normal kidney. The cortex and medulla are well proportioned and well differentiated. The cortex does not bulge markedly beyond the cut edge of the capsule. The cortical markings are distinct, especially the vascular apparatus, in which the Malpighian bodies are unusually prominent, standing out as minute bright red points. Microscopically, the vascular apparatus throughout is deeply engorged. The inter-tubular capillaries are widened and tortuous, and the red cells in them are closely packed and show marked effect of reciprocal pressure, or are even fused. The epithelium in the convoluted tubules is in a state of advanced granular degeneration.

"Note—The histological changes in this kidney correspond entirely with those occasionally encountered in athletes, who, after severe exertion, have suddenly subjected the overheated body to the effects of cold, in which event contraction of the peripheral vessels is followed apparently by loss of vasomoter control in the kidneys. The vessels dilate and become tortuous and the red cells in them fuse. At the same time, stagnation of blood results in nutritional changes in the lining epithelium of the tubules and granular degeneration occurs. Very similar changes are met with in the kidney in subjects dead of tetanus, of hydrophobia, or of certain irritant poisons. The condition is relatively rare, but by no means unknown, as a sequence of simple ether anaesthesia and some-

times follows nephrectomy of the opposite kidney. In the latter circumstance, the combination of anaesthesia and suddenly increased functional demands upon the remaining kidney consequent upon the removal of its fellow is possibly the best available explanation. Death usually succeeds upon complete anuria and may occur within a few hours or be delayed for days; thus in one patient death occurred on the twenty-first day after an operation for epithelioma of the penis."

This would seem to be an instance most damaging to the reputation of the test. Had the pathologist discovered chronic lesions of the "good" kidney, we should have interpreted it so. But the congestion was surely of recent origin and doubtless did not exist before operation. A test of prophetic value is beyond our fondest hope.

A mild kidney infection (shall we call it pyelitis) may interfere little or not at all with renal function as far as we can determine it. Rowntree and Geraghty cite such an example, and from No. 31 of our series, with pus and staphylococci from both ureters, we recovered 63.+% in two hours.

Instances illustrating the effect of general anaesthesia are too few in our series to warrant definite statements yet it would seem that general anaesthesia does not interfere with subsequent excretion of phenolsulphonephthalein

Our conclusions agree quite closely with those of Rowntree and Geraghty, but we venture the following criticisms:

I. The intake of phenolsulphonephthalein may indeed be measured more accurately than that of the constituents of urea, and its output is more prompt and more readily measurable than that of phloridzin or indigo carmin. Yet, while we have found it far superior to the other artificial color tests, it shows marked superiority to the estimation of urea percentage, and urea in cgm., and especially the experimental polyuria test, only as an index of the total kidney function, not of the relative function of the two kidneys as compared with each other.

In 12 out of 15 ureter catheter examinations in which the data justified a comparison of the phenolsulphonephthalein output with the urea in cgm., these two indicators told the same tale and gave the same ratio of functional ability for the two kidneys, while the three cases in which they differed were better diagnosed by comparison of successive specimens obtained by ureter catheter than by any evidence derived from single specimens. Moreover, the study of successive specimens prevents errors from eccentricities of urinary excretion during the first half hour of ureter catheterization.

II. We have found that in 5 out of 11 cases ureter catheterism so diminished the output of phenolsulphonephthalein (proved by subsequent tests) as to render it most misleading in determining total kidney capacity; although, as we have already observed, the relative inefficiency of the diseased kidney, as compared with its mate, was correctly indicated by the test.

Hence we deem it advisable usually to make two phenolsulphonephthalein tests, one with ureter catheterism, one without, just as one would make two urea tests, one with ureter catheterism, and one on a twenty-four hour specimen of urine. If the output of the drug is high during ureteral catheterization, clearly the second test is superfluous for estimating functional ability at this time.

When the phenolsulphonephthalein test is employed with the ureter catheter only for the purpose of comparing the relative efficiency of the two kidneys, the patient's discomfort may be lessened, the possibility of error by extra-catheter flow diminished, and much time saved by collecting urines for successive brief periods, and comparing these with each other. As a general rule, for phenolsulphonephthalein, as for urea readings, 20 to 30 minute periods are preferable to shorter ones.

The microscopic findings remain, as ever, the most important elements in ureter catheter diagnosis. Urea and phenolsulphonephthalein estimations should confirm these, and successive specimens, for comparative readings are unnecessary in the majority of cases, but are most helpful in the precise interpretation of ambiguous ones.

That phenolsulphonephthalein is not to be depended upon as an absolute or infallible guide of the actual renal function, or of the reserve force of the kidneys to withstand the shock of nephrectomy, any more than any other test, is suggested by the three casualties cited, in one of which nephrectomy resulted in death by kidney insufficiency despite a good showing before operation, and in two of which such death did not result in spite of a falling output.

Finally, we must once again insist that we agree entirely with the essential parts of the report of Drs. Rowntree and Geraghty. We have felt obliged to insist upon the points of difference rather than upon those of agreement between our findings and those recorded in their publication. In our hands, to be sure, the test has not been found mathematically accurate, but that is only because of the unfathomed human element, both in our patients and in ourselves. We regard it the equal of any test yet devised for comparing the functional value of the two kidneys, and superior to any for determining the total renal capacity. This latter phase of its use is the important one, affording a tangible basis for estimating renal function. Obviously the power of the kidneys to rid the blood of one drug should not be assumed to be an indicator of their ability to eliminate all other substances. Yet in practice, the excretion of the drug under consideration has been an amazingly accurate index of renal efficiency. Just how low the output may fall before the danger point is reached in a given situation should not be fixed dogmatically. No two cases are alike in all particulars.

We shall continue to use the phenolsulphonephthalein test, not only as a help in pre-operative diagnosis, but also in many other connections.

REFERENCES.

1. Journal of Pharmacology and Exp. Therap. July, 1910. Vol. I, No. 6.

Table I.

Test Readings on Duboscq Colorimeter.

Each specimen contains 25 cc. of deep amber urine plus the amount of a solution of 0.6 gram of phenolsulphonephthalein to 1000 cc. distilled water, required to make the percentages given. This same aqueous solution was used as a standard for comparison.

Actual Percentage	Immediate Reading	Reading 48 Hrs. Later	Reading same date as last column, after adding 1 drop of blood to 15 cc. of mixture	Reading of original mixtures, one month later
50	53.7	53.2	50.0	trace
25	25.5	21.4	18.0	no color
10	10.2	3.0(?)	...	no color
5	5.17	ft. trace	...	no color
3	3.35	no color	...	no color
2	2.5	no color	...	no color
1	trace	no color	...	no color
½	ft. trace	no color	...	no color

(Table II, See Next Page)

Table III.

Showing Time of Maximum Excretion.

(Intramuscular Method of Injection

Patients with normal kidneys—each giving over 60% in 2 hours.

Case No.	Percentage 1st ½ hour	Percentage 2nd ½ hour	Percentage 3rd ½ hour.	Percentage 4th ½ hour
38	35.2	20.7	8.0	4.0
39	32.9	18.7	7.4	4.0
40	31.6	19.2	9.9	5.3
31	29.4	14.0	13.9	5.7
Average	32.3	18.2	9.8	4.8

TABLE II

Cases with Supposed Normal Kidneys

Case No.	Age	Date	Diagnosis	Time of appearance in minutes	First Hour: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Second Hour: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Total % P. for 2 hrs.	Remarks
2	34	5-26-10	Syphilis Spinal Cord	8	57.9–440–0.4	13.3–191–0.5	71.2	Partial retention. Third hour, 4.1–90.
		5-31-10		9	45.9–340–0.8	10.1–105–0.85	56.0	Third hour, 0.8–30–?; fourth hour, trace–23. No change of condition, clinically.
4	26	6-2-10	Recent gonorrhoeal epidid. Temperature below 100° for past week	7½	45.9–690–0.1	12.5–150–0.4	58.4	Third hour, trace–32; fourth hour, trace–60.
5	42	6-2-10	Lacerated scrotum	5½	45.8–375–0.4	9.6–195–0.6	55.4	Third hour, trace–138; fourth hour, tr.–112.
6	40	6-2-10	Swelling of testicle for 12 yrs.	8	41.9–135–1.5	10.9–98–1.2	52.8	Third hour, 4.3–138; fourth hour, tr.–60.
9	29	8-25-10	Convalescent from epididymitis and acute prostatitis	10	38.2–450–	16.7–332–	54.9	Leaving hospital today.
10	24	8-25-10	Hypospodias. 2½ wks. post op.	12	51.0–430–	5.0–235–	56.0	
		9-15-10	Excell't condit'n. Before 2d op.	11	35.7–365–	13.7–120–	49.4	
		10-18-10	" " " "	7	55.6–400–	6.2–66–	61.8	
		10-20-10	" " " "	5	56.0–215–	trace–80–	56.+	Intravenous injection.
21	30	10-15-10	Gonorrhoeal Epididymitis	7½	48.0–43–	6.6–37–	54.6	10 days after operation. Temp. below 100°. Patient up and about.
		10-20-10		5	53.1–70–	trace–65–	53.+	Intravenous injection.

TABLE II—CONTINUED

82	35	3-30-11	Syphilis of testicle	9	51.5–62–	7.7–110	59.2	3 weeks after intramuscular injection of Salvarsan.
47	30	1-4-11	Pain and restricted motion of L. hip	10 10	R. ureter–14.5–18–2.5 L. ureter–10.3–13–2 8 Bladder–trace–140–?			X-ray shadow near, but not in L. ureter. Urines normal.
93	25	4-19-11	R. renal colic (?) for two weeks; no pain now	20 14	R. ureter–1.0–6–1.2 (¼ hr.) L. ureter–0 6–5–1.3 (¼ hr) –1.4–3–? (6 min.) Bladder–1.0–3–?			Urines normal.
96	32	4-30-11	Cystitis	10 14	R.U.–2.4–18.–.28 (½ hr.) L U.–4.7–30.–.25 (½ hr.) Bl.–9.5–94–? (½ hr.)			Urines from kidneys normal.
100	45	5-3-11	Chronic Prostatitis	10 10	R.U.–3.7–5–? (½ hr.) L.U.–3.6–4.5–? (½ hr.) Bl.–? (½ hr.)			Urines from kidneys normal.
105	?	5-11-11	Kidneys normal	no color in 20 m.	R.U.–sl. tr.–40– L.U.–sl. tr.–45– Bl.– tr.– 10– } 1 hour from injection			Urines from kidneys normal.

TABLE IV

MEDICAL CASES

CASE No.	AGE	DATE	DIAGNOSIS	TIME OF APPEARANCE IN MINUTES	FIRST HOUR	SECOND HOUR	TOTAL % P. FOR 2 HOURS	REMARKS
					{% of drug excreted} - {amt. urine collected in cc} - {% of urea}	{% of drug excreted} - {amt. urine collected in cc} - {% of urea}		
29	75	12-3-10	Cardiac	13	35.2-30-	8.3-25-	43.5	Mitral Insufficiency. Very large heart. Marked oedema of lower extremities. Urine contained considerable albumin, hyaline and granular casts. Blood pressure varied from 70 to 100 mm. Hg. At time of test, almost in extremis, unconscious, Cheyne-Stokes respiration. Died in a few days. No autopsy.
84	45	4-1-11	Cardiac	10	34.0-25-	12.9-29-	46.9	Mitral Insuff. Large heart. Moderate oedema of legs. Orthopnoea. Blood pressure 170. Urine—trace albumin, no casts, Sp. Gv. 1025; 36 ℥ in 24 hours, urea 2.6%. Died April 6. No autopsy.
87	53	4-4-11	Cardiac	17	41.7-120-	17.4-80-	59.1	Very large heart; mitral insuff.; slight oedema of legs; moderate dyspnoea. Temp. not over 100°. Urine—much alb., no casts, 1028. Bld. press. 125.
88	63	4-4-11	Cardiac	8	30.1-110-	13.6-190-	43.7	Fairly large heart; marked oedema. Urine—1020, much alb., hyaline and granular casts, urea 1.6%. Blood pressure, 145.
85	65	4-1-11	Cardiac Nephritic Autopsy	11	14.5-30-	8.0-14-	22.5	Fairly large heart; marked oedema; orthopnoea. Urine—1020, trace alb., few gran. casts; ℥ 35 in 24 hrs., urea 1.8%. Blood pressure 155. See text for necropsy findings.
43	27	12-27-10	Parenchym. Nephritis	15	13.9-66-	14.7-60-	28.6	No symptoms. Urine—considerable alb., hyal. granular and epithel. casts. This urinary condition known to have existed six years.

TABLE IV—CONTINUED

49	35	1-7-11	Parenchym. Nephritis	11	37 3-100-	7.4-80-	44.7	Feet swell. Sometimes vomiting and giddiness. Urine—980 cc in 24 hrs., 1.8% urea, much albumin, hyaline and granular casts.
60	22	2-1-11	Bilateral haematuria Par. Nephr. (?)	?	R. ureter-1.5-2-0.2 (2nd ½ hr.) L. ureter-3.7-7-0.2 (2nd ½ hr.) Bladder-?			Cystoscopy under ether anaesthesia. Urine—much albumin and casts.
102	35	5-5-11	Parench. Nephr. (?)	20 20	R. U.-2.0-3- (20 min.) L. U.-6.2-9- (20 min.) Bladder-?			Very alcoholic patient. Had one profuse haematuria. Kidneys not palpable Urine—albumin and hyaline and granular casts, no pus.
58	?	1-28-11	Pregnant 4 m. Pathological urine	9	35.6-110-	23 8-125	59.4	No headache, no nausea, no oedema. Urine—albumin and hyaline, granular and blood casts. Subsequently cleared.
103	?	5-7-11	Pregnant 5½ m. Pathological urine	6	40.0-145-	11.5-54-	51.5	Urine contains alb. and casts. No grave symptoms.
64	67	2-7-11	Interstitial Nephritis	9	10.4-180-	4.0-120-	14.4	Occasional haematuria. General condition excellent at times of all 3 tests.
		3-16 11	Ulcer of bladder	8 8	R.U.-1.5-50-0.7(½ hr.); 1.8-55-0.8(1½ hr.) L.U.-13-65-0.8 (½ hr.); 1.3-43-0.7 (½ hr)	4.5-170-	10.4	Very sl. oedema of feet. Urine—trace albumin, little pus and blood, no casts found.
		3-21-11		9	4.5-110-	3.0-60-	7.5	
80	57	3-30-11	Interstitial Nephritis ?	?	14.7-138-	18.9-227- Third hour-; tr.-130-	33.6	Oedema of legs. Dyspnoea. Large heart. Urine 116 to 144 $\bar{3}$ in 24 hours daily for many weeks; 1010, trace albumin, few gran. casts. Blood pressure 185.
81	45	3-30-11	Interstitial Nephritis	5	8.3-260-	7.0-230-	15.3	Sl. oedema heretofore, none now; easily dyspnoeic. Moderately enlarged heart. Albuminuric retinitis, Urine—35 to 64 $\bar{3}$ in 24 hrs.; 1010, much albumin, no casts. Blood pressure 240

TABLE IV—Concluded

Case No.	Age	Date	Diagnosis	Time of appearance in minutes	First Hour: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Second Hour: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Total % P. for 2 hours	Remarks
77	42	3-24-11	Interstitial? Nephritis? Hepatic Cirrhosis	15	3 1–15–	8 3–40–1 8	11.4	Typical picture of cirrhosis. Thin man with large belly. Been repeatedly tapped. Urine—25-40 ℥ in 24 hrs.; 1015, much alb., few casts, urea 1.2%. Blood pressure 110 to 135.
56	30	1-24-11	Polycystic Kidneys	46 38	R. U.–tr.–20–1.0 L. U.–tr.–20–1.1 Bl –	R.U.–tr.–11–1.5 L.U –tr.–20–1.2 Bl.–tr.–95–0.9 (at end of 2nd hour)	tra's +	Was in good general health when last seen, 2 months later.
62	36	2-2-11	Polycystic Kidneys	10 10	R.U -3.7-50-0.5 (½ hr.); 3.3-90-0.3 (½ hr.) L U.-2.1-28-0.5 (½ hr.); 0.9-28-0.2(½ hr.) Bl -tr.-116-0.3 (end of 1 hr.)	tr –140–0.3	10. +	No symptoms.
		2-3-11		10	38 5–40–1.9	17 9–27–1.65	56 4	R kidney later removed. Normal convalescence.
69	?	2-22-11	L haematuria	6	55.6–102–1.6	9.1–114–1.45	64.7	Blood seen coming from L. ureteral orifice L. nephrectomy stopped haematuria—kidney seemed normal. Urine—no pus, no casts.
78	53	3-25-11	Alcoholism Arterio-scleros's Terminal pneumonia	8	45.5–30–	8.7–22–2.4	54.2	Was having temperature 102° to 103° daily. Irrational at times Symptoms of "wet brain." No oedema. Heart not enlarged Urine—35 ℥ in 24 hrs.; trace albumin, urea 0.9%, granular casts. Blood pressure 125. Died April 6. No autopsy.

TABLE V

Miscellaneous Cases

Case No.	Age	Date	Diagnosis	Time of appearance in urine	First Hour: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Second Hour: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Total % P. for 2 hours	Remarks
101	?	5-4-11	Ureteral Calculus	9	43.5–105–	16.0–105–	59.5	Stone 2 inches from pelvis of kidney. Urine not infected. Stone removed by operation, after test. Convalescence normal.
34	50	12-9-10	Calculus in bladder	10	14.2–32–1.6; 27.2–25–2.3 (½ hr.) (½ hr.)	10.4–165–0.9	51.8	Pus in urine. Residual urine ʒi. Sharp cystitis. Operation after test. Convalescence normal.
70	67	2-27-11	Calculus in bladder	8	31.6–51–	10.1–60–	41.7	Few hyaline and granular casts. No stone in kidneys. Bladder stone and middle lobe of prostate removed suprapubically. Convalescence normal.
71	20	2-28-11	Bladder tumor	8	14.4–140–	20.8–250–	35.2	Bilateral nephrotomy showed normal kidneys. Convalescence normal.
38	42	12-20-10	Tuberculosis, lungs, sem. vesicles, epididymis	5	35.2–34–; 20.7–26– (½ hr.) (½ hr.)	8.0–22–; 4.0–26– (½ hr.) (½ hr.)	68.0	Few pus cells in urine. Kidneys apparently normal. Some vesicles and epididymis removed. Convalescence normal. Living but weakening from pulmonary tuberculosis, 6 months later.
63	41	2-7-11	Tuberculosis, testicles, prostate, lungs	8			44.6	Ill 8 months. Lost 50 pounds. Few red blood corpuscles in urine; No casts, no pus. Some frequency of urination. Daily temperature up to 103°. No operation.
33	57	12-8-10	Stricture of urethra	8	23.15–35–; 14.0–26– (½ hr.) (½ hr.)	16.4–66–	53.5	Perineal section for stricture Dec. 1, 1910. Normal temperature now.

TABLE V—CONTINUED

Case No.	Age	Date	Diagnosis	Time of appearance in urine	First Hour	Second Hour	Total % P. for 2 hours	Remarks
					{% of drug excreted} – {amt. urine collected in cc} – {% of urea}	{% of drug excreted} – {amt. urine collected in cc} – {% of urea}		
52	55	1-10-11	Stricture of urethra	18	trace–210–	13.9–270–	14.0+	Neither test is accurate, as patient was not catheterized and he had residual urine.
		1-28-11		none allowed	trace–92–(1 hr. from injection)	15.6–88–	16.0+	Resection of stricture Jan. 10, 1911. Had several cardiac attacks, in one of which he died about three weeks after operation. No autopsy.
55	30	1-17-11	Stricture of urethra	none allowed	18.0–15–(1 hr. from injection)	20.8–32–	38.8	Test immediately after perineal section. Much blood in urine.
		1-28-11		none allowed	31.3–90–(1 hr. from injection)	18.5–16–	49.8	
107	43	5-23-11	Stricture of urethra	9	42.1–43–	15.8–30–	57.9	Emergency operation for perineal abscess and stricture 16 days ago. Has been mildly delirious with septic temperature. Better now; temperature below 100°. No oedema. No headache. No hiccough. Later developed femoral thrombosis.
32	46	12-8-11	Perineal abscess, prostatic?	7	33.3–260–; 10.0–152– (½ hr.) (½ hr.)	6.9–204–	50.2	Perineal section, Nov. 24. Now walking about. Residual urine 5i.
39	35	12-20-11	Perineal abscess, prostatic?	6	32.9–42–; 18.66–48– (½ hr.) (½ hr.)	7.4–42–; 4.0–46– (½ hr.) (½ hr.)	62.9	Operation 32 days ago (perineal section).
40	40	12-20-11	Perineal abscess prostatic?	7	31.6–25–; 19.2–26– (½ hr.) (½ hr.)	9.9–27–; 5.3–25– (½ hr.) (½ hr.)	66.0	Prostatic abscess drained 6 days ago.

TABLE V—Concluded

50	30	1-7-11	Prostatic abscess; Acute retention	10 10	R.U.-8.3 55-0.7; 8.3-60 0.9 L.U.-2.9-32-0.3; trace-18-0.25 Bl.-? (½ hr.) (½ hr.)	6.2–150–0.3 trace–55–0.3	25.8+	Acute retention 11 days ago; was catheterized every four hours. Abscess broke into bladder, as noted by cystoscope. No casts nor pus from ureters; no pus in bladder urine now.
		1-13-11		10	18–50–1.4	31.3–65–1.3	49.3	First and second hour specimens perhaps confused. Urine clear. Steady general improvement.
1	49	5-26-10	Double Hydrocele, infected	12	27.5–215–0.6	14.6–112–1.0	42.1	Third hour:—5.5.-88-1.4. Operation 3 weeks ago. Temperature normal. Wound suppurating. General condition good.
		6-7-10		13	7.94–353–0.1; 8 84–83–0.55 (½ hr.) (½ hr.)	6.4–128–0.3; 4.7–86–0.6 (½ hr.) (½ hr.)	27.9	Third hour:—4.0-150-0.5.
53	23	1-14-11	Diagnosis? Pus in urine Fever prolonged	10	37.0–61–1.4	31 4–240–1.0	68.4	Urine—Trace albumin, few hyaline casts, few red blood cells, pus. Note large amt. urine in second hour—Bladder probably not completely emptied at end of first hour.
		1-?-11		13 9	R.U.-5.0-5-2.5 (½ hr.); 6.9-6.5- (½ hr.) L.U.-6.4-7-3.3 (½ hr.); 5.0-4.5- (½ hr.) Bl.-13.6-120-0.75			Urine from both kidneys normal—Bladder urine contains pus but no casts.
51	55	1-10-11	Malignant papilloma of ant. urethra	10	22.7–265–	12.8–255–	35.5	Test made 3 weeks after operation for supposed stricture. Condition fair. Later stood emasculation well.
65	16	2-16-11	Exstrophy of bladder	9–	22.1–34– ; 16.0–48– (½ hr.) (½ hr.)			Urine collected in Kelly pad for one hour only; some of it lost. Pt. later stood Peter's operation well. Doing well 3 months after operation.

TABLE VI.

Hypertrophy and Carcinoma of Prostate.

Case No.	Age	Date	Diagnosis	Time of Appearance in Minutes.	First Hour {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Second Hour {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Total % P. for 2 hours.	Remarks
7	68	6-7-10	Hypertrophy	11	26.5–53–1.8	4.6–3.3–1.5	31.1	Third hour—:2.55-51-1.6. Perineal prostatectomy May 12, 1910. Now, residual ℥ i. Urine—1010, albumin (trace), hyaline and granular casts.
8	74	8-8-10	Hypertrophy	12	31.2–62–	16.4–64–	47.6	Led catheter life past 20 years. Kidneys free from infection 8 weeks after operation. Bladder badly infected.
		8-16-10		10 Intravenous	19.2–55–	5.2–85–	24.4	Intravenous injection.
		10-1-10		13	19.3–115–	4.0–70–	23.3	Perineal prostatectomy August 18, 1910.
		10-12-10		10	R. N. trace–192–0.34			Satisfactory convalescence.
				10	L. N. trace–108–0.35			Readings made 3 days after test. Colors had faded notably. Patient in good condition June, 1911.
11	57	8-25-10	Hypertrophy L. Pyonephrosis	9	27.8–332–	14.2–215–	42.0	Perineal prostatectomy Sept., 1909. Now pain over bladder, vomiting, headaches. No residual.
		9-10-10		15	R. – 5.0–110– (1 hr.)			Urine from bladder thought to come largely from R. kidney. Condition better.
				15	Bl.–13.0–185–			
		9-29-10		?	Bl.– 8.33–?–1.75 (¾ hr.)			Better condition.
				0	L. – 0–?–0.85			
15	60	9-15-10	Carcinoma ?	12	13.2–140–	12.5–140–	25.7	Residual urine ℥ i. Pus in urine. Frequency of urination 4 years. No operation.
16	50	9-15-10	Carcinoma ?	14	21.7–165–	11.6–118–	33.3	Symptoms 2 months. Complete retention now. Fever. Pus in urine.
17	63	10-1-10	Carcinoma	17	7.8–39–	5.5–14–	13.3	Complete retention.
		10-18-10		16	14.7–33–	9.0–45–	23.7	Suprapubic drainage under cocaine Oct. 14. Died (apparently of cancerous cachexia) in Dec., 1910. No autopsy.

TABLE VI—CONTINUED

22	55	11-6-10	Hypertrophy	45		4.0–150–	5.5	Complete retention. Bad condition. See text.
								Nov. 4—1450 cc urine in 24 hrs., urea 0.9%, much albumin.
		11-11-10		50	2.5–50–	5.5+–65–	8.0+	Nov. 11—3500 cc urine in 24 hrs., urea 1.1%. Suprapubic drainage, Nov. 8th.
		12-9-10		25	good trace–100–0.8	16.6–95–1.0	17.0+	Perineal prostatectomy Nov. 15th. Pt. at work 6 mos. later.
24	65	11-11-10	Hypertrophy	30	trace–36–?(½ hr.); trace–31–?(½ hr.)	trace–50–	traces	Urine 1850 cc in 24 hours with 0.9% urea. Suprapubic drainage under cocaine before the test. Very feeble. Perineal prostatectomy under spinal anaesthesia after test. Died in 7 days. See text. No autopsy.
25	67	11-22-10	Hypertrophy	10	21.0–84–	8.3–34–	29.3	Median bar. Complete retention one wk. Urine 360 cc with 1.0% urea in 12 hours. Perineal drainage Nov. 23.
		11-29-10		20				Specimens stood 3 days; then not measureable. Chetwood cautery operation without anaesthesia Dec. 2.
		12-3-10		11	14.7–35–?(½ hr.); 15.6–43–?(½ hr.)	16.1–62–	46.4	Satisfactory convalescence.
26	40	11-23-10	Hypertrophy	10	19.2–20–?(½ hr.); 7.4–12–?(½ hr.)	22.7–126–	49.3	Urinary symptoms 20 months. Perineal prostatectomy Nov. 23.
		12-3-10		8	33.8–65–?(½ hr.); 16.6–82–?(½ hr.)	9.0–130–	59.4	Easy convalescence.
46	65	1-4-11	Hypertrophy	10	R.U.–17.9–64–?(½ hr.); 3.1–9.0–?(½ hr.)			Residual urine six ounces; almost constant dribbling. No operation.
				10	L.U.–4.0–10–?(½ hr.); 7.1–18–?(½ hr.)			
					Bl.–?			
79	70	3 27-11	Hypertrophy	16	17.4–85–	12.4–65–	29.8	Catheter life for years. General condition good. Urine—900 cc with 1.6% urea in 24 hrs.; pus and hyaline casts. Suprapubic prostatectomy under spinal anaesthesia Mar. 29. Vomited 3 days thereafter; thence convalescence satisfactory.
		5-13-11		9	22.7–46–	13.9–40–	36.6	On April 6, 930 cc urine, 3.1% urea.

TABLE VI—Concluded

Case No.	Age	Date	Diagnosis.	Time of Appearance in Minutes.	First Hour {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Second Hour {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Total % P. for 2 hours.	Remarks
91	59	4-16-11	Hypertrophy	12	29.1–72–	13.2–50–	42.3	Urinary symptoms 6 years; now catheter life. Urine, 1012; trace alb.; blood, pus, and hyaline casts. Operation after test. Easy recovery; no sign of uraemia.
94	80	4-21-11	Hypertrophy	18	faint trace–50–	3.0–26–	3.0+	Urinary symptoms 17 years. Suprapubic prostatectomy and removal of bladder stone, under gas and ether, against advice, after test. Death on 5th day of uraemia and pneumonia. See text.
95	79	4-21-11	Hypertrophy	60	11.4–62–	19.5–78–	30.9	Perineal drainage 4 wks. ago. Perineal prostatectomy 3 wks. ago. Now in bad condition generally (toxic). Still large residual.
		4-30-11		90	8.9–58–1.8	13.6–70–2.2	22.5	Mentally clearer; tongue clean.
		5-4-11		85	10.4–80–	10.4–70–	20.8	Been worse since above; now better.
		5-8-11		90	25 5–122–	15.6–63–	41.1	Better.
		5-13-11		75	8.9–81–	17.4–80–	26.3	General condition much worse. Sp. Gv. 1004; urea 0.7%.
97	66	5-2-11	Hypertrophy	12	35.0–70–	28.4–76–	63.4	A few ounces of residual urine several years. Chetwood cautery operation 4 years ago, relieving complete retention. Urine infected. General health excellent.
98	65	5-3 11	Hypertrophy	11	47.6–90–	17.9–59–	65.5	Residual urine two ounces, infected. Perineal drainage after test. Voided 35 ounces before operation, 100 ounces (in 24 hrs.) after operation. Good recovery.

TABLE VII

COMPARISON OF PHENOLSULPHONEPHTHALEIN AND UREA OUTPUTS

CASE NO.	DIAGNOSIS	% OF P. 1ST HR.	% OF P. 2ND HR.	UREA %	TOTAL UREA IN 24 HOURS IN GRAMS	REMARKS
49	Chr. Parenchym. Nephritis	37.3	7.4	1.8	17.6	
77	Cirrhosis Liver	3.1	8.3	1.2	12.6	Emaciated. Abdomen repeatedly tapped.
81	Chr. Interstitial Nephritis	8.3	7.0	1.0	15.9	
84	Cardiac	34.0	12.9	2.6	28.0	Bad condition. Died 5 days later.
85	Cardiac	14.5	8.0	1.8	18.9	In bad condition. Died next day.
22	Prostatic Hypertrophy	1.5 2.5	4.0 5.5	0.9 1.1	13.1 38.5	Tests 5 days apart. Suprapubic cystostomy between tests.
24	Prostatic Hypertrophy	trace	trace	0.9	16.6	Very poor condition. Perineal prostatectomy after test; death in 7 days.
79	Prostatic Hypertrophy	17.4	12.4	1.6	14.4	Good condition. Survived subsequent prostatectomy.
45	Pyonephrosis	38.4	16.6	1.0	16.0	Good condition. Subsequent nephrectomy. Death in 53 hours. See text.

TABLE VIII

Renal Tuberculosis

Case No.	Age	Date	Diagnosis. Organs Infected	Time of appearance in urine	First Hour: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Total % P. for 1st hr.	Second Hour: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Total % P. for 2 hours	Remarks
3	30	5-31-10	Bladder R. kidney (removed)	8	34 8–139–0.55	34.8	10.2–53–1.35	45.0	R. Nephrectomy April 14, 1910. Daily tem. to 100°. Voiding 40 ounces urine daily.
		6-7-10		10	14.7–53–1.5(½ hr.); 8.2–49–1.0(½ hr.)	22.9	2.8–45–0.9(½ hr.); 3.6–43–1.6	29.3	In 3rd hour, 0.7% P.; 4th hour trace. Readings 30 hours after collection. General condition apparently as good as May 31st. In 3rd hour, 3.0% P.
36	?	12-14-10	After nephrectomy	11	30.1–45–	30.1	10.4–106–	40.5	L. kidney removed July 14, 1910. Now few pus cells in urine. Has gained 26 pounds.
37	17	12-15-10	R. kidney	40	R. U.–tr.–10–0.4 (½ hr.)				
			Bladder	?	Bl.–28.6–?–1.5 (70 m. fr. injection)				
		1-?-11		?	R. U. (no color in 20 min.)				R. U.—urea 0.05%; pus, no casts.
				10	L. U. (not measured)				L. U.—urea 0.7%; hyaline and granular casts, few blood cells. Second test under ether anaesthesia.
41	32	12-21-10	Kidney ? Testicle	10	38.5–170–	38.5	8.3–200–	46.8	Urine, 1009, 0.4% urea, trace albumin, pus, red blood cells.
42	?	12-21-10	R. kidney L. kidney (?) Prostate;	11	33.3–76–	33.3	11.9–52–	45.2	Urine (bladder) —0.8% albumin (bulk), pus, few red blood cells, no casts. Much pus from R.; little from L. kidney.
		4-11-11	vesicles;	0?	R. V. 0–				
			L. epididymis	15	L. V. 8.0–8–?(½ hr.); S.3–20–?(½ hr.)	16.3	trace–12–? (both kidneys)	16.3+	
		4-13-11	Elbow	15?	trace–46–	trace	14.2–50–	14.2+	15 min. delay allowed (not tested).
		5-16-11		?	41.7–96–	41.7	17.4–62–	59.1	R. Nephrectomy April 15, 1911. Satisfactory convalescence.
44	45	1-3-11	Remaining kidney. Genital and pulm. Tbc.	11	9.4–44–	9.4	10.4–51–	19.8	R. Nephrectomy 1 yr. ago. Urea, 1.1%.
66	36	2-16-11	L. kidney Bladder	7	R. U.–11.4 16–1.2(½ h.); 10.1–12–1.6(½ h.)	36.3	13.9–50 (both kidneys)	50.2	From R. kidney—no pus, cocci and bacilli (not acid-fast).
				12	Bl.–6.6–28–1.1(½ h.); 8.2–12–1.4(½ h.)				From L. kidney—pus.

TABLE VIII—Concluded

72	?	3-4-11	L. kidney ?		R.U.-ft.tr.-25-0.(½ h.); f.tr.-12-0.2(½ h.)	traces	good trace–36–0.3 { both kidneys }	traces	From R. kidney—no pus
			Bladder		L.U.-ft.tr.-73.0(½ h.); f.tr.-34-0.25(½ h.)				From L. kidney—little pus + bacilli not acid fast.
					Bl.-0-59-0(½ hr.); f.tr.-12-0.2(½ h.)				From Bladder—pus + many acid-fast bacilli.
									Test under spinal anaesthesia.
		3-5-11		8	44.6–80–1.2	44.6	6.6–84–0.8	51.2	Guinea-pig killed by sediment of bladder urine.
74	35	3-6-11	Remaining kidney	10	16.1–52–1.4	16.1	10.2–50–1.5	26.3	L. Nephrectomy 18 mos. ago. Now pus, tubercle bacilli, and hyaline and granular casts from R. kidney. Voids q i h.
75	27	3-8-11	L. kidney	8	R.U.-3.1.8-?(½ h.); 5.6-11-?(½ h.)	10.4 +	10.4–40–? { both kidneys }	20.8 +	Delirium Tremens preceding test. Hematuria 3 mos. ago and for past 7 days.
				9	L.U.-trace-8-?(½ h.); 1.7-12-?(½ h.)				Urine from R. kidney – casts and few pus cells.
					Bl.-?				Urine from L. kidney—no pus.
		3-14-11		12	20.5–58–	20.5	8.9–77–	29.4	On Mar. 15, R. kidney explored, found hypertrophied; L., nephrectomy (large old focus; small active focus).
		4-8-11		16	32.9–70–	32.9	5 2–40–	38.1	Satisfactory convalescence.
76	36	3-14-11	Both kidneys Epididymis	17	Bl.-ft.tr. 15-?(½ hr.); ft.tr.;-20-?(½ h.)	12.0+	15.0–70–	27.0	R. testicle removed 5 yrs. ago.
				8	L.U.-5.3-12-?(½ hr.); 6.7-15-?(½ h.)				R. kidney and L. epidid. removed after test. No uremia.
									Infection of kidney wound. Primary union of scrotum.
									Recovery.
104	26	5-11-11	L. kidney Bladder	9–	R.U.–15.0–10–?(½ h.)		11.1–22–? { 1½ hours; both kidneys }	57.1	L. Nephrectomy May 16. Easy convalescence.
				?–	Bl.–31.0–23–?(½ h.)				
		5-23-11		5					
		5-25-11		3 (intravenous)	52.3–105–	53.3	20.6–42–	73.9	Good condition.
					18.6–60–?; 31.1–42–?; 8.0–75–?; 3.8–135–? (quarter hour intervals)	61.5	3.0–105–?; trace–100–? (half hour intervals)	64.5	Good condition.

TABLE IX

Surgical Renal Disease—Not Proved Tubercular

Case No.	Age	Date	Diagnosis	Time of appearance in urine	First Hour: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Total % P. for 1st hr.	Second Hour: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	Total % P. for 2 hrs.	Remarks
12	35	9-9-10	Bacilluria fr. R. kidney	18 18	R.U.-8.8-15-1.2(½ hr.); L.U.-1.5-5-1.4(½ hr.); Bl.-? (½ hr.); 14.0-60-? {at end of 1 hour}	24.3			No pus, but bacilli from R. kidney. L. kidney normal. Probably much extra-catheter flow from L. side.
13	52	9-9-10	Infect'n both kidn's; Tbc.?	16	22.7-410-	22.7	11.4-260-	34.1	Poor condition clinically.
14	28	9-10-10	R. renal infection; Tbc.?	0 20	R.U.-(no color in 45 min.) L.U.-2.8-5.5-? (25 min.) Bl.-?				Probably tubercular. Urinary frequency only symptom. In same interval of time R. ureter gave 20 cc urine with 0.55% urea; L. ureter, 4 cc with 3.2% urea.
19	30	9-20-10	L. renal infection	17 19	R.U.-4.8-10-?(¼ hr.); 5.1-12-?(¼ hr.) L.U.-1.8-13-?(¼ hr.); 3.0-12-?(¼ hr.) Bl.-1.7-10-? (end of ½ hr.)				Pus and colon (?) bacilli from L. kidney; R. normal. In other specimens, in same interval of time, R. kidney gave 0.53 cgm. urea and L. kidney 0.33.
23	24	11-1-10	Stone and infection, R. kidney	6 6	Bl.-ft. trace-44-? (½ hr.) L.U.-18.9-195-? (½ hr.)		17.9-210-? {both kidneys; 1½ hours}	37.0+	R. nephrectomy later. Good recovery.
27	33	12-2-10	L. acute pyelonephrosis	30	5.0-60-? (½ hr.); 5.0-50-? (½ hr.)	10.0	10.0-105-?	20.0	Bladder stone removed 6 wks. ago. High temperature, sometimes reaching 106°, since operation. Much pus in urine. Voids 45 ounces urine with 1.0% urea in 24 hours.
		12-8-10		18	7.1-53-? (½ hr.); 8.3-55-? (½ hr.)	15.4	10.0-90-?	25.4	Temperature lower. Horse-shoe kidney found at operation Dec. 12; died Dec. 20, low temp., hiccoughs, vomiting.

TABLE IX—CONTINUED

30	36	12-8-10	Sl. infection both kidneys	9	R.U.-2.0-43-0.5(½ h.); 2.3-20-0.7 (½ h.)	16.6 +			Little pus and colon bacilli from both kidneys.
				9	L.U -2 2-52-0.4(½ h.); 4.0-25-0.4 (½ h.)				
					Bl - trace-25-? (end of 1 hr.)				
		12-12-10		9	25 0-55-? (½ h.), 15.4-65-? (½ h.)	40.4	5.3-35-? (½ hr.); 3.3-65-? (½ hr.)	49.0	
31	25	12-8-10	Double renal infection	11	29.4-110-? (½ h.); 14.0-50-? (½ h.)	43.4	13.9-?-? (½ hr.); 5.7-92-? (½ hr.)	63.0	Pus and staphylococci from both kidneys
		12-10-10		?	R.U.-16 2-28-? (½ h.)				Probably extra-catheter flow from L. side.
				?	L.U.-5.9-15-? (½ h)				Other specimens gave equal urea % from kidneys.
					Bl -?				No operation.
35	37	12-13-10	R. pyonephrosis (?)	11	12.5-23-? (½ h.); 13.4-42-? (½ h.)	25.9	17.0-68-?	42.9	Pus and blood in urine for 5 yrs.
		1-13-11		45	Bl.-5.2-40.0-0.3	5.2+	21.4-54-0.9	29.6 +	From behavior of catheter flow, most of bladder color thought to come from L. kidney.
				12	L.U.-trace-7-0.7		3.0-7-1.5		
45	50	1-13-11	R. pyonephrosis	13	38.4-74-	38.4	16.6-96-	55.0	Had had Rt. renal colic. Voided 1600 cc urine in 24 hrs.—1% urea, 0.5% (bulk) albumin, much pus. On Jan. 14, R. nephrectomy. Death 53 hrs. later (see text). At autopsy, marked vascular engorgement of remaining kidney.
48	35	1-5-11	Double renal infection	14	R.U.-4.8-35-0 8(½ h.); 2 9-17-1.4 (½ h.)	19.3	5.0-39-?	30 5	Frequency of urination; had haematuria, Tuberculosis suspected—marked improvement on tuberculin.
				14	Bl.-4 5-45-1.0 (½ h.); 7.1-55-0.9 (½ h.)		6.2-65-?		
		2-9-11		11	32.5-92-	32.5	10.1-58-	42.6	Condition much better.
57	39	1-24-11	R. renal calculus	16	Bl.-1.1-50-0.15 (½ h.); 7.1-80-0.15 (½ h.)	17.0	12.0-50-1.4 (both kidneys)	29.0	Bladder urine—trace albumin and pus. No pus from L. kidney.
				10	L.U.-3.8-85-0.15(½ h.); 5.0-31-1.9(½ h.)				
		3-18-11		?	43.1-202-	43.1	12.5-214-	55.6	Later pyelotomy, followed by renal sepsis, then R. nephrectomy. Thereafter few casts in urine.

TABLE IX—CONTINUED

SURGICAL RENAL DISEASE—NOT PROVED TUBERCULAR

CASE NO.	AGE	DATE	DIAGNOSIS	TIME OF APPEARANCE IN URINE	FIRST HOUR: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	TOTAL % P. FOR 1ST HR.	SECOND HOUR: {% of drug excreted} – {amt. urine collected in cc} – {% of urea}	TOTAL % P. FOR 2 HRS.	REMARKS
59	35	1-31-11	Rt. renal infection	0	R.U.-no color in ½ hr.	19.0 +			R. kidney—pus and colon (?) bacilli.
				10	L.U.-19-15-? (½ hr.)				L. kidney—bacilli but no pus.
					Bl.-trace-9-0.02 (end of ½ hr.)				Radiograph negative.
		2-10-11		11	R.U.-1.9-17-? (½ hr.); 0.9-27-? (½ hr.)				From other specimens, urea % of Rt. kidney 0.5; of L. kidney 1.5.
				5	L.U.-19.2-50-? (½ hr.); 10.6-100-? (½ hr.)	37.7	11.0-?-?	48.7	
					Bl.-5.1-44-? (end of 1 hr.)				
61		1-31-11	Infected polycystic kidney	σ in 30 min.	Bl.-tr.-20-				Pus from R. kidney; now from L.
				10	L.U.-tr.-20-	traces			Later R. nephrectomy; good recovery (see text).
67	43	2-21-11	L. Renal calculus	13	10.4-50-	10.4	11.4-75-	21.8	Has pulmonary tuberculosis. Very alcoholic. Urine contains much pus and mucus.
		2-23-11		7	R.U.-2.8-?-?; 2.0-6-1.7; 4.2-10-1.0; 3.8-9-1.2 (quarter hour intervals)	17.4	6.7-23-1.9	35.3	Two quarter-hour intervals of bladder specimen correspond in time to last two of R. U. specimens. Bladder specimens believed to have obtained color chiefly from R. kidney.
				35	Bl.-3.8-20-0.3; 0.8-3-0.5 (¼ hr.) (¼ hr.)		11.2-90-1.0 (1 hr.)		
		2-27-11		10	7.0-10-?	7.0	10.4-80-?	17.4	L. nephrectomy on Mar. 2.
		3-16-11		15	19.2-74-?	19.2			Patient weak from pulmonary tuberculosis. Died 16 days later from same (see text).
68	38	2-21-11	Infected R. moveable kidney	12	16.4-32-	16.4	32.3-428-	48.7	Catheterized at end of each hour by nurse; apparently bladder was not emptied at end of first hour. Patient stood subsequent nephropexy nicely. Doing well 4 mos. later.

TABLE IX—Concluded

73	33	3-5-11	R. renal focal suppuration	8	13.6-60-	13.6	18.0-60-	31.6	Chills past 10 days; temp. 105° past 48 hrs. Nephrectomy after test. Sepsis followed, but no evidence of renal insufficiency. Final result good.
86	31	4-8-11	L. renal infection Old cystitis	14 12	R.U.-2.7-3.5-3.6 (20 min.) L.U.-tr.-5.0-1.6 (20 min.)		32.0-60-? {both kidneys / 1 hr. 40 min.}	37.5+	Recent acute L. renal pain and infection. Test made under chloroform.
		4-15-11		Bl.-2.8-7.0?- (20 min.) 10 26.3-370-		26.3	17.9-131-?	44.2	Condition better. No fever. No anaesthesia.
		5-2-11		9-	R.U.-5.4-10-1.1; 5-12-0.9; 7.1-11-0.6				
				10- 10-	L.U.-3.2-6.2-0.6; 0.6-2-?; 0.7-1.5-? Bl.-21-9-0.3; 6.2-10.2-0.6; 6.5-13-0.3 (20 min. intervals)	36.8	19.2-?-? (both kidneys)	56.0	Chloroform anaesthesia. L. ureter catheter worked poorly. Acute symptoms entirely gone. Infection of L. kidney practically cleared up.
92	36	4-18-11	R. renal infection and stone	13 10-	R.U.-1.1-4.5-? (¼ h.); 1.7-9.5-? (¼ h.); L.U.-5.2-7.5-? (¼ h.); 13.9-22.-? (¼ h.); Bl.-16.1-60-? (at end of 1 h.)	38.0			Other specimens gave urea, 0.7% from R. kidney, 2.0% from L.
108	65	5-25-11	R. renal infection and stone	13	30.0-105-	30.0	23.3-138-	53.3	Excellent general condition. Refused operation.
110	45	5-29-11	Infection (?) remaining kidney	?	41.9-105-	41.0	15.6-50-	56.6	One kidney removed 7 yrs. ago. Now pyuria and pain over remaining kidney.

LIGATION OF THE INTERNAL ILIAC ARTERIES*

for

1.—Hemostasis in extensive extirpation of pelvic viscera for cancer.

2.—To prevent recurrence of apparently completely extirpated cancers of the pelvic viscera.

3.—To retard the growth of inoperative cancers of the same viscera.

4.—To arrest hemorrhage in inoperable cancers, chiefly of the uterus.

With Report of Six Cases.

IRVING S. HAYNES, PH.B., M.D.

Uterine cancer is the most insidious and fatal disease by which women are attacked.

Three out of every four cases coming under observation, in Germany, are absolutely inoperable and beyond all hope of cure; the percentage is much higher in this country.

Of the cases subjected to operation by various surgeons of international reputation we find the following figures hold. Regarding operation by the abdomen according to the so-called Wertheim method (although this was practiced and described by Werder, and was modified from the older methods of Ried, Strumpf and Clark).

Operability of Cases of Uterine Cancer.

Operator.	Cases.	Inoperable.	Operated.
Hocheisen	1,706	1,538	168
Schindler	588	471	117
Zurhelle	253	168	85
Stauda	156	52	104
Hannes	361	216	145
Doederlein	151	78	73
Wertheim	400	232	168
Totals	3,615	2,755	860

Percentage of operable cases, 23.8.

Combining the statistics of Wertheim, Hannes, and Schindler we find that their average mortality was 22.92% and their absolute cures according to the formula of Werner is 14.06%. In a similar manner considering the statistics of the simpler vaginal operation as carried out by Schuchard, Schauta, Standa, Hannes, Doederlein, Glockner, Zurhelle and Olhausen we find that the average mortality is 10.64% and the absolute cures after five years after Werner's formula is 16.75%.

Operations, Cures According to Weiner's Formula.

Abdominal route.	Primary mortality.	Cures.
Wertheim	22.50%	24.7 %
Hannes	32.6 %	14.3 %
Schindler	13.67%	3.18%
Average	22.92%	14.06%
Vaginal operations.		
Schuchard	9.6 %	20. %
Schauta	10.8 %	12.6 %
Hannes	8. %	28.8 %
Standa	20. %	23. %
Doederlein	16.4 %	15.8 %
Glockner	8.46%	9.72%
Zurhelle	4. %	14. %
Oldhausen	7.7 %	10. %
Average, vaginal	10.64%	16.75%
Average, abdominal	22.92%	14.06%

Difference in favor of the vaginal route, 12.28% less mortality, primary.
Greater percentage of cures, 2.69%.

The radical operation formerly performed by Wertheim is not necessary. The reason is that recurrence was thought to be in the pelvic lymph nodes, hence their removal was obligatory. Further experience shows this is not true, much to our surprise. In four out of five cases of recurrence the growth re-

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turns in the vaginal scar. In one case out of 104 did a recurrence take place in an iliac lymph node and in only two cases in the inguinal glands.

It is absolutely impossible for anyone to determine during the operation whether the lymph nodes are involved or not. If not involved their removal is not necessary, if they are involved their removal is useless for there has been involvement beyond the field of possible removal.

Schauta, who made most extensive investigations along this line, concluded that in 43.3% there was no lymphatic involvement, hence the operation (the extreme radical) was unnecessary; in 43.3% the glands were so extensively involved beyond the field of possible removal that the operation was useless; and in only 13.3% were the accessible nodes involved. But the attempt to extirpate these few possibly involved nodes is not justifiable when there is an immediate mortality of the extremely radical operation of 66 to 72%. For these reasons Wertheim and other leading surgeons have abandoned this extreme operation.

Indeed the reaction has been so marked that some of the former radical advocates have concluded that as good permanent results can be obtained by the so-called palliative measures, as a thorough curettement followed by local applications of the actual cautery, zinc-chloride, fuming nitric acid, formalin and probably best of all the acetone treatment advocated by Gellhorn.

Close analysis of these palliative measures gives one a real surprise. We are so accustomed to think that nothing but wide excision in cancer alone gives any hope that the figures offered by these various operators cause us to sit up and take notice. Chrobak treated 408 cases with fuming nitric acid. His patient lived from 3 to 5 years, one even for 22 years and one for 20. The primary mortality is probably not more than that due to the anaesthetic plus 1 or 2% from peritonitis following perforation of the uterus by the curette. These palliative measures are used by Lomer, Mond, Roessling, Fleischmann, Zacharias, A. Reeves Jackson, Chrobak, Webster, Gellhorn and others. Czerney has recently revived this treatment and it has received the favorable commendation of Murphy.

The preceding statements are based upon a comprehensive article by Dr. John B. Murphy and Dr. Frank W. Lynch in the VIII volume of the American Practice of Surgery by Bryant and Buck.

Inasmuch as hemorrhage is one of the frequent accompaniments of the actual cautery method, and as we know that starvation of the cancerous bearing tissues is a potent factor in not only aiding a cure but also in preventing a return of the growth I am led to advocate the ligation of both internal iliac arteries as a preliminary step in the treatment of these cases with completion of the operation by a thorough curettage and the use of the acetone treatment after the method of Gellhorn. Ligation of the internal iliac produces definite anemia of three distinct pelvic systems. (Byron Robinson, Annals of Surgery, Vol. 35, p. 189). 1. The lower part of the ureters, the bladder (and in the male the prostate), and the urethra. 2. The uterus and its adnexa, the vagina and the vulva. 3. The lower portion of the rectum and anus. I would therefore advocate the use of this ligation in the following conditions:

1. All inoperative pelvic cancers attended with hemorrhage, profuse and recurring, more especially from the uterus.

2. As a preliminary procedure to prevent hemorrhage and recurrence in all such operations as extirpation of the bladder, prostate and urethra; hysterectomy with or without removal of the upper portion of the vagina; excision of the rectum and anus, or vulva.

3. As a preliminary step in the so-called palliative operations upon the uterus and vagina, in connection with the later technic of Gellhorn.

I should claim that it was a wise procedure in all cases of suspected or actual malignant pelvic growths.

If the suspected case ultimately proves to be positive it will tend to delay if not actually prevent a return. In the actual case it serves to arrest hemorrhage and delay growth.

The comfort, rapidity and ease with which extensive pelvic operations can be performed would in itself justify the performance of this operation as a preliminary step in many instances. This contention has been fully demonstrated in my own cases and especially by one very recently, April 27, in the practice of Dr. Furniss at the Red Cross Hospital. At the doctor's request I assisted him in the operation of ligation of both internal iliacs preliminary to total extirpation of the bladder in a woman. There was none of the very active bleeding and troublesome oozing. We would have had a practically bloodless field had we ligated both ovarian arteries. Their free anastomosis with the uterine vessels furnished the only vessels we had to ligate. A running catgut suture in the other tissues effectually controlled all the others.

The effect of the ligation upon uterine hemorrhages in the otherwise inoperable cases is positive and immediate. How long such arrest is maintained I am unable to state.

The ligation of the arteries, so far as my experience goes, is attended with no bad effects, immediate or remote. The only complaint has been of a heavy tired feeling, which was present before the operation, in one case that lasted for about two weeks.

Ligation of the internal iliacs was first performed by Dr. W. Stevens of Santa Cruz, in 1812. (Keyes, Bryant and Buck, Vol. IV, p. 508).

Pryor (Am. Jour. of Obs., June, 1896) records 34 cases of malignant disease of the uterus treated by ligation of the internal iliac arteries, with one death.

Bainbridge (Woman's Med. Jour., April 1911) reviews the subject of arterial ligation for various conditions and especially for irremovable cancer of the pelvic organs.

Ligation of the internal iliacs by the transperitoneal route is usually an easy operation. Difficulties may arise from the filling up of the pelvis by the growth or from its extension to the pelvic tissues covering the arteries, or from a low course of a very tortuous external iliac artery. The internal iliac is found by incising the peritoneum in line with the course of the artery, and below the level of the pelvic brim at a distance of an inch and a half from the mid-line. The ureter will be seen attached to the under surface of the outer flap of the peritoneum; it must not be disturbed. The artery is covered by the pelvic fascia which needs to be divided in the line of the peritoneal incision. Blunt dissection exposes the artery and a double ligature of No. 3 plain gut is passed by the carrier from within out, being careful to lift the artery away from the vein while insinuating the ligature carrier beneath the artery. Before tying the ligature, have some one get the femoral pulse, if it is not arrested by traction on the ligature, tie the latter and close up the peritoneal incision by a running catgut suture. On the left side the mesentery of the sigmoid may add a little to the operative work. I have found it best to firmly draw the sigmoid to the left and proceed as before. Keyes (ibid) mentions gangrene of the limb, peritonitis and secondary hemorrhage, as the chief dangers. If sepsis is present or follows, such possibilities might occur. But one would not perform the operation in septic pelvic conditions and sepsis is such a remote contingency in previously clean cases that I consider such dangers too remote to be seriously considered. So far as my experience goes the operaton has been followed by no bad effects,

immediate or remote. Pryor in 34 cases of malignant disease of the uterus, had one death.

Kelley (Operative Gynecology, Vol. II, p. 331) testifies to the value of this procedure in such conditions as the following:

"When there is much lateral infiltration the embarrassment from the hemorrhage in cutting through the infiltrated tissues is sometimes so great that the operator has to abandon all ideas of operative relief, and finish the operation the best way he can. I operated upon a case of this kind. As the operation proceeded, it was found impossible to extirpate the disease in the broad ligaments and to check the free oozing from the diseased tissue which was cut; in order, therefore, to control the entire blood supply of the part, I ligated both internal iliac arteries. After the ligation all pulsation in the pelvis on both sides ceased. The patient made a good recovery and suffered in no way from the artificial pelvic anemia, and the disease returned so slowly that she lived over two years after the operation."

I have operated in the following six cases without operative mortality, with such freedom from hemorrhage as to be of the greatest value to the patient and assistance to the operator in rapidity and ease of the work.

From my experience, I would further advise that in all cases where the genital or urinary tracts are involved and after ligation of the internal iliacs that both ovarian arteries should likewise be tied whether extirpation of the organs is contemplated or not. The free anastomosis of the ovarian with the uterine furnishes too much blood to the latter and unless ligated before division would interfere with the operation.

Case I.—A. G., German, aged 38. Admitted to Harlem Hospital, Feb. 24, 1908. Discharged, March 24, 1908.

Family history.—Negative.

Past history.—Has had 3 children. For the past 5 weeks has lost weight rapidly. Feels she is growing weaker. Has pain over the lower part of the abdomen.

Physical examination.—Is a medium sized woman, thin, anemic and cachectic.

Abdomen is retracted, scaphoid, tense but not rigid. No masses can be felt.

Vaginal. Perineum is relaxed, cervix hard and the seat of a large ulcerating cauliflower growth. The uterus is enlarged and fixed in position. No masses can be felt in the fornices. T. 98. P. 92. R. 18.

Urine, Feb. 28, showed trace of albumin, otherwise negative. Feb. 29, operation.

The upper third of the vagina was dissected free of the bladder and the rectum. In performing this separation the base of the bladder was opened into. This was sutured and healed promptly, causing no subsequent trouble.

The abdomen was then opened, both internal iliacs ligated with No. 3 plain gut at a point about an inch below the bifurcation of the common arteries. The uterus with the appendages and broad ligaments was removed. The peritoneum was closed transversely in the pelvis. The ventral wound closed in layers. Iodoform gauze strips were placed into the vagina.

By March 4 the gauze packing was all out. Primary union took place in the abdominal wound. The patient was out of bed on March 18.

The highest T. P. R. occurred one day following the operation and were 101, 120, 26.

March 24, discharged. T. 99½, P. 78. Apparently perfectly healed. This extensive extirpation was rapidly carried out as I did not have to stop for troublesome hemorrhage and all oozing was easily controlled at last by continuous gut sutures.

Final result.—Patient died about six months later from local return of growth.

Case II.—Mrs. L. W. Private patient referred to me by Dr. Neff. Stout woman of 60. Married. Admitted to the Red Cross Hospital, June 1. Discharged, June 20, 1910.

Complains of very severe pain in the back over the sacrum.

Married 22 years, never been pregnant.

Three weeks ago began to have sharp pain in the left hip. Hemorrhage from the vagina for the past 24 days.

Physical examination negative except for the pelvic region. The uterus is generally enlarged, more especially on the right side. It is quite firmly fixed by an induration extending more especially into the right broad ligament but some into the left side. The cervix is not enlarged, and there seems to be no vaginal or rectal involvement.

Positive diagnosis not ventured.

Operation.—June 2. Curette removed masses of tissue unmistakably cancerous.

Abdomen opened. Uterus so far involved that the cancerous tissue showed through the peritoneum. Both broad ligaments extensively infiltrated. Radical operation impossible, therefore the internal iliacs were ligated with No. 3 plain gut and the wound closed in layer sutures.

Highest T. P. R on the day following the operation, 101, 100 and 28. Wound healed by primary union. Sat up in bed on the 10th day and was out of bed on the 11th day. She went home on the 18th day. In spite of this extensive carcinoma this patient recovered, the growth was held in check and she did her own housework for over a year after the operation and only died March 4, last, 1912.

Case III.—Mrs. I. C., private patient, 45 years of age, married, has one child 17 years old. In the summer of 1910 she flowed freely for 5 weeks. She was curetted and the uterine scrapings submitted to one of the best laboratories in the city. The diagnosis then was adenocarcinoma. She was so discouraged and disheartened that she did nothing for herself, expecting to die very soon. However as a year passed without death having come, and as she was in constant pain she determined to seek relief, one way or the other. She consulted me April 4, 1911. Thorax and abdomen were negative. No glands palpable. The uterus was slightly enlarged, very tender, freely movable. The cervix was red but not eroded. There was a thin watery non-irritating vaginal discharge.

I operated at the Red Cross Hospital, April 7, accepting the laboratory diagnosis, and believing I had a very slow growing cancer to deal with.

Both internal iliacs were easily ligated and a total removal of the uterus, upper part of the vagina, tubes and ovaries carried out. The appendix was also taken out.

The pelvic condition was extremely interesting. Especially in view of the laboratory diagnosis under which I was working. There was a small amount of pale straw fluid in the pelvic cavity. All the blood vessels leading into and from the pelvic viscera were deeply injected. A more "angry" looking condition I have never seen. This appearance confirmed, in my mind the correctness of the previous diagnosis. However an examination of the specimen removed showed that the uterus was the seat of a general fibroid change, without any evidence of cancer.

The patient was out of bed on the 10th day and left the hospital at the end of three weeks. Of course she has remained cured until the present time.

Case IV.—G. B. Admitted to Harlem Hospital, Nov. 17, 1911. Discharged, Feb. 7, 1912. Widow aged 48.

Last August, after sexual intercourse, first noticed a burning and frequent urination. Slight discharge. In October noticed that the vaginal orifice was hard, contracted and tender, especially on the left side. She enters complaining of pain in the back and frequent and painful micturition.

Physical—Thin anemic nervous woman. Inguinal glands are palpable and very hard. Nothing found in the abdomen or chest. There is a hard indurated mass involving the anterior part of the vaginal orifice, urethra and clitoris, larger on the left than right, but on both sides firmly attached to the ischiopubic rami. Gonococci found in the vaginal discharge.

W. B. C. 17000, Polys, 70%.

T. P. R. normal.

Nov. 25. Both internal iliacs were ligated with No. 3 plain gut in continuity. There seemed to be no pelvic growth apparent. The abdominal wound was closed and the entire vulva with the clitoris, the urethra up to the vesical sphincter and the lower half of the anterior wall of the vagina was excised.

Pathological examination showed that the growth was an adenocarcinoma originating from the galnds of Bartholin on both sides. The T. P. R. the day after the operation were 101, 102 and 24. There were primary union in the abdominal wound and quick healing in the perineal gap. Patient was out of bed on Dec. 16.

There was a return of the growth in the shape of a small nodule at the left of the vesical sphincter. This was enucleated and thoroughly cauterized on Jan. 4 by electric cautery.

At the time of her discharge, Feb. 7, 1912, there was no recurrent growth apparent.

She was placed in a home for incurables. At the present, April 29, I am informed by her sister that there is a nodular recurrence at the site of the original operation which has begun to ulcerate. She is in a very debilitated condition.

Case V.—M. G., Bohemian, 53 years old, married, entered Harlem Hospital, Jan. 22, discharged Feb. 6, 1912.

Patient had good health up to 6 months ago when she was taken with a profuse leucorrhea, occasionally blood stained. Her menstrual history began at 14, was always irregular and painful. Has had two children, the last 22 years ago.

Sept. 8 she was admitted to one of the large city hospitals and some vaginal operation performed, its nature was not explained to her. She was in the hospital 13 days. Three weeks ago she again went to the same hospital, was there for a week and discharged with nothing operative having been done.

Her present complaint is excessive bleeding with constant pain.

Examination shows a large uterus and cervix, deeply indurated; the vaginal walls, bladder and rectum all being involved. There are irregular hard nodules in and about the cervix. Diagnosis made of inoperable uterine cancer.

January 23. Both internal iliacs were tied with No. 3 plain gut in continuity. There was some difficulty experienced in finding the left artery as the uterus was much enlarged and had become firmly attached to the sigmoid and the mesentery of the gut was greatly thickened and shortened.

In order to make sure of the artery about which the ligature was passed it was necessary to have an assistant feel for the femoral pulse. Twice was the ligature placed about the external iliac, which from a low bifurcation of the common iliac and a low course of the external iliac simulated the position of the internal. This precaution, however, served to finally secure the right artery.

The wound healed by primary union.

The highest T. P. R. on the day following the operation, were 101 4-5, 124, 28.

No reaction whatever. Patient out of bed on Feb. 2, eleven days after the operation. She left the hospital on the 6th of Feb. There were no more hemorrhages from the uterus and the patient said she felt greatly relieved.

Case VI.—Mrs. G. K. Private patient. Referred to Dr. Upton. Large, stout German woman of 46.

Menstruation regular and painless. Has had 2 children and 6 misses. Last pregnancy 10 years ago.

For the past month has had a vaginal discharge of blood, coming in a regular gush and lasting for an hour or two, every day. Last menstruation was 4 or 5 months ago and was normal. No trouble with bowels or urine. Examination is negative except for pelvis. There is an excavating ulceration in the cervix which has disappeared. There is apparently no infiltration present. The uterus is generally enlarged and freely movable without pain. No glands enlarged.

Diagnosis of cervical carcinoma in an early stage made. A total hysterectomy after ligation of both internal iliacs with removal of tubes, ovaries and upper part of the vagina made. The appendix was also removed. April 19 at the Red Cross Hospital examination of the specimen shows that the cervical growth was benign papilloma.

The patient is making satisfactory recovery. The condition not being proved malignant really does not interest us, except to demonstrate that the operation of ligating the internal iliac arteries was easily performed and has been attended with no unfavorable result.

The subsequent history showed that the clinical diagnosis was correct. Recurrence of the cancer took place in the vaginal scar and progressed very rapidly, terminating in the death of the patient during the past summer.

REFERENCES.

1. Presented at a meeting of the Eastern Medical Society, May 10, 1912.

THE DIAGNOSIS OF FOREIGN BODIES IN THE ALIMENTARY AND RESPIRATORY TRACTS OF CHILDREN.*

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There are few conditions in children which are more confusing and puzzling than those sometimes caused by the presence of a foreign body in the alimentary or respiratory tract. It would appear at first thought that there is no reason for any perplexity, the history furnishing the diagnosis. Experience demonstrates, however, that the matter is not so simple, for several reasons: Firstly, the history is often indefinite and misleading; secondly, the primary symptoms because of their transitory nature are either overlooked or not considered as important evidence; and, thirdly, the alimentary and respiratory tracts tolerate a foreign substance for a long time before giving any manifest reaction.

Even though the history be definite and the primary symptoms well defined still the quiescent period which frequently follows tends to allay the suspicion of the entry of a foreign body and the entire occurrence may in time be entirely

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forgotten and receive no consideration in the estimation of a later affection which may be the direct result of the existence of a foreign object within one of the tracts.

Thus it is that many cases present themselves for diagnosis, exhibiting certain clinical features which, conforming in their ensemble to acknowledged entities are so considered, until the extrusion or a belated intrascopic examination reveals a foreign substance as the cause of the condition.

The diagnosis in children, in whom this accident occurs far more frequently than in adults, is thus attended with many difficulties; and close observation and keen analysis of the objective and subjective data are necessary in order to arrive at such a mental attitude as will suggest radiographic and endoscopic examinations in order to verify or exclude the tentative diagnosis of a foreign substance.

Classification.

Because of their infinite variety, the substances which have been ingested can be classified only in an imperfect manner. Such a classification, however crude, is nevertheless necessary since much depends upon the nature, shape and size of the object. The substances may first be divided into inorganic and organic. The former may be subdivided into smooth, round bodies (coins, beads, pebbles) and irregular sharp bodies (nails, pins, glass). The organic substances may be divided into vegetable substances which swell in the presence of fluids (bean), vegetable substances which do not swell in the presence of fluids, and animal substances. This classification, modified from that suggested by Hoffman, covers the vast majority of such substances as enter the organism of the child. Such a vegetable substance as a bean, though harmless in the alimentary tract, may work havoc in the air passages because of its obstructive capabilities due to its power of swelling in the presence of fluids. The objects which usually cause trouble in the alimentary tract are metallic, either smooth or sharp, as coins, pins, nails, etc. The bodies most frequently found in the bronchi of children appear to be nails, pins, buttons, beans and beads; in the esophagus—coins. Pins and needles give the greatest trouble in the stomach and intestine. A smooth, rounded substance will not cause an ulcerative condition as rapidly as an irregular sharp one. Though usually single, occasionally more than one body may be ingested or inspired.

History.

A sane adult may be able, as a rule, to give a clear and definite history of the ingestion or inhalation of a foreign body. Such a history is, however, seldom to be elicited even from older children because of the fright and emotion attendant upon the event. Occurring as it usually does, when no eye witness is present, it is often difficult to determine the nature of the object the child had in its hand at the moment of the accident. Even an apparently positive statement, by those interested, of the occurrence of the accident should not be given absolute credence, for many are the recorded cases, where with such an affirmative history and even an accompanying story of a spasm of suffocation, the foreign body was later found outside of the patient, in the bed or within the pocket of an attendant. Willard (1) cites a case he observed in the practise of one of the prominent surgeons, of a child who was tracheotomized and prolonged search made for a foreign body supposed to have been inspired, but which was later found within a pocket of the child's clothing. Closer examination disclosed that the dyspneic symptoms were due to double lobar pneumonia.

Nor is it always possible to tell from a mere consideration of the history alone, whether the foreign body was swallowed, or what is of more serious im-

portance, inhaled. Tracheotomy has not infrequently been performed in search for a body in the bronchi, which was ultimately passed per rectum.

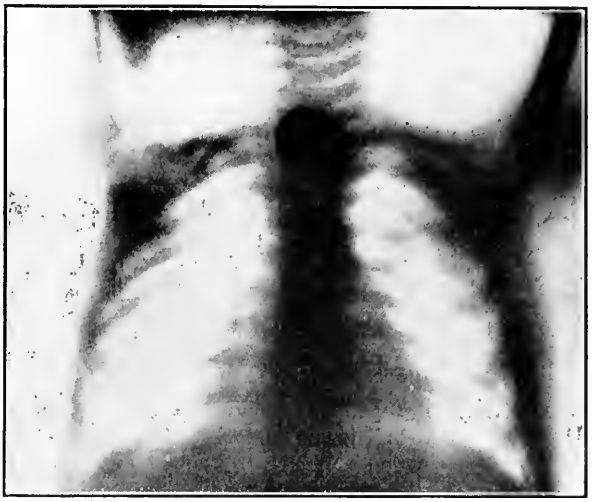

Fig. 1.—Case 1. Coin (quarter) in esophagus, at point of cervical constriction.

It may be stated, therefore, in dogmatic fashion that in order to avoid confusion, the history should be completely subordinated in the preparation of the diagnosis, and that in neither its positive or negative aspects should it be allowed to unduly influence the final conclusion. If at all considered the patient should be given the benefit of the doubt, in the sense that the benefit of a thorough examination be conferred and careful surveillance, both immediate and subsequent, be instituted.

Symptomatology.

The study of the symptomatology necessitates, first, the consideration of the symptoms manifested at the time of and immediately following the ingestion of a foreign body, which are designated as the primary symptoms and may comprise the sum total of the eventual history of the case, and secondly, the consideration of the later symptoms manifested as a result of its continued presence in the organism. The primary symptoms, mild or severe, transitory or prolonged, may be separated from the onset of the secondary by a period during which no reaction is apparent. In other words, after the distress resulting from the passage and entry of the body has subsided, there follows a period of calm, of variable duration, during which the foreign body lies as a non-irritant mass. This toleration is a variable factor and depends in a general way both upon the nature of the invading substance and upon its location; it being essential for tolerance that the substance neither markedly obstruct the lumen of the tube within which it rests, nor irritate its walls. The intestinal tract, because of its almost constant contact in its functional activity with foreign bodies, exhibits great tolerance to their presence, and similarly an object which because of its size and situation does not interfere with the entrance of air into the lungs will be tolerated for a long time even in the air passage.

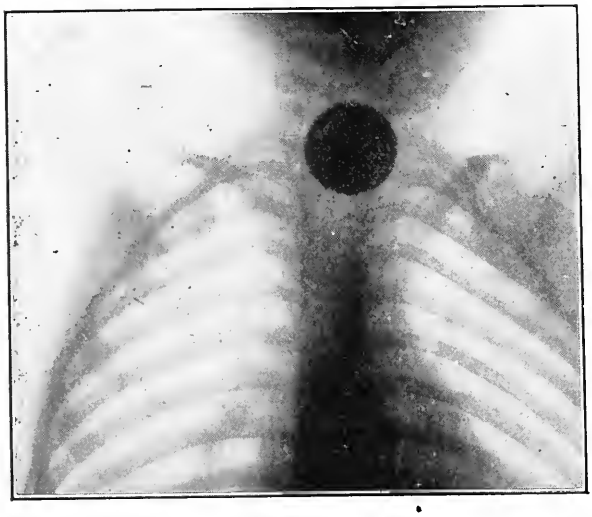

Fig. 2.—Case II. Coin (quarter) in esophagus, at point of cervical constriction.

Foreign Bodies in the Alimentary Tract.
The Esophagus.

Primary Symptoms.—The swallowing of a foreign body and its passage into the esophagus is attended by no alarming symptoms if it be small and smooth. A large or sharp object causing greater irritation will induce a voluntary effort to expel it. This may be futile and then the centripetal action of the musculature of the pharnyx, acting in spite of the voluntary expulsive effort will cause it to pass rapidly downward. Depending upon its size and shape, it may pass either directly into the stomach or become impacted in the esophagus. In its passage there may be induced a temporary spasm of the glottis, resulting in severe inspiratory dyspnea, cyanosis, slow heart action and collapse. These symptoms, designated as the initial paroxysm, are reflex, due to irritation of the superior laryngeal nerve and as Crile has proved experimentally, they occur only as a result of irritation of that portion of the esophagus opposite the larynx. This initial paroxysm of suffocation is of variable severity, often assumes a threatening aspect and may even result in death. The alarming symptoms may partially subside, continuing in a milder form, or completely subside, giving way to a period of calm, as the body passes the sensitive area.

Impaction is naturally most likely to occur at those particular points where, either through intrinsic or extrinsic causes, the lumen of the tube is narrowed. This obtains usually at three points. First, at the beginning of the esophagus opposite the sixth cervical intervertebral cartilage and behind the cricoid cartilage; second, in its thoracic part opposite the fourth dorsal vertebra behind the aortic arch and at the point where it is crossed by the left bronchus; third, at its termination where it passes through the diaphragm. As a result of anomalous construction, other sites of constriction may exist, Mehnert having described thirteen. A pathological stricture may be the cause of impaction of a foreign body which might otherwise pass through. Billroth performed esophagotomy on a child for the removal of a metallic button which had become arrested by a stricture caused by the ingestion of a solution of potash a year previously. Foreign bodies are most frequently impacted at the cervical and least frequently at the diaphragmatic constriction.

Once a body is impacted it usually remains fixed in that particular position because both of muscular spasm and of swelling of the mucous membrane. As a rule, the higher up the impaction occurs, the more marked are the primary symptoms and the more rapid is their onset. It is important to remember, however, that except for some slight difficulty in swallowing there may be for a long time no symptoms resulting from the presence of a foreign body impacted in any part of the esophagus.

Pain, dysphagia, regurgitation, nausea, vomiting, singultus, cough, hoarseness and hemorrhage are symptoms common to impaction in any part of the esophagus. The prominence of one symptom or another is associated with impaction in some particular situation.

Impaction high up, if the foreign body be sharp, often presents a clinical picture strongly suggestive of the condition. The appearance of the child with its head held rigid and drawn to one side, the mouth open and dribbling, asso-

Fig. 3.—Case III. Coin (cent) in esophagus, at point of cervical constriction.

ciated with the immediate and increasing dysphagia and early regurgitation is rather characteristic of the presence of such a foreign body in this situation. If the object is dull or smooth this picture is duplicated later when ulceration due to prolonged pressure appears. The pain is referred to the throat; it is constant and increased during swallowing, while tenderness may be elicited either by backward pressure on the cricoid cartilage or bilateral compression of the soft parts opposite this point. Vomiting is not common, but when it does occur it usually follows the hoarse, paroxysmal cough. As examples of impaction at this location the following cases are reported:

Case I. Samuel K., aged six years, was presented with the history that he had swallowed a silver "quarter" seven days before. Immediately after the accident the child coughed and choked considerably and there was much nausea. Four hours later he was examined by a physician, who allayed the fears of the parents and assured them that the body had passed into the stomach and would in due time appear in the stools. At this time and for the two following days

there were no symptoms except that the child complained of slight pain in the neck. Then difficulty in swallowing of solids appeared. The pain soon became worse and dysphagia rapidly increased in severity, so that on the fifth day even fluids were regurgitated. Upon the sixth day a bougie was passed into the stomach without meeting any obstruction. Physical examination was negative except that the head was held to the left side and its movements were restricted. Regurgitation of solids was immediate and deglutition very painful. Fluoroscopic examination showed the coin in the cervical portion of the esophagus (Fig. 1). It was removed by the aid of Graefe's basket or the "coin catcher" of Dupuytren, as the instrument is sometimes called, though not without some difficulty. The child vomited after extraction, but the recovery was prompt and without sequelae.

Case II. Florence B., aged 3½ years, while playing with a silver "quarter" suddenly began to choke and cough, and then fainted. The mother ran to the child, and suspecting the cause of the paroxysm inserted her finger in the child's throat and thought she felt the coin which slipped from her finger when she attempted extraction. Soon after this the child recovered, became quiet and appeared comfortable. Five days later difficulty in the swallowing of solids was noticed, but there was no regurgitation or vomiting. She sought medical advice. After examination of the throat, a catheter was passed into the stomach without much difficulty and assurance was given that the coin had passed out of the esophagus and would make its exit in the normal way. Complete relief of symptoms followed for 14 days, when gradually increasing difficulty in the swallowing, even of fluids, regurgitation, a slight cough and general depression, prompted the mother to again seek advice. General physical and local examination did not yield any data. Fluoroscopic examination showed the presence of a coin in the esophagus in the segment of cervical constriction. After a radiograph (Fig. 2) had been made, the coin was removed without any difficulty by means of a "coin catcher."

Case III. Fanny C., aged 2 years, was presented with the history that for the preceding ten days the child had been croupy and had considerable difficulty in swallowing. Examination showed the pharynx and tonsils to be in normal condition. There was considerable drooling of saliva, and the mouth was kept open. Its mucous membrane was normal.

Unable to account for the condition on any other basis than the existence of some obstruction in the esophagus, a fluoroscopic examination was made which disclosed a coin (Fig. 3) in the cervical constriction. There was no history of its ingestion.

Case IV. Warren T., aged 9 months, was presented with the history that on the previous day the child had a paroxysm of dyspnea and coughing. The parents suspected that a safety-pin had been swallowed. The next day the child vomited considerably, did not take its nourishment and the cough persisted. The child was examined by Dr. Henry Koplik, who diagnosed a pin in the esophagus. The radiograph (Fig. 4) corroborated this diagnosis and showed the pin in cervical part of esophagus. It was removed with aid of the esophagoscope.

A great variety of foreign bodies has been removed from this situation; Rigby (2) extracted a toy bicycle; Mayo (3) a buckle, etc.

The symptoms present as a result of impaction of the foreign body at the point of aortic constriction are usually mild. The dysphagia is not severe, the pain being slight and referred to the midscapular region. Cough is an early and persistent symptom. Bodies are well tolerated in this situation. In the majority of cases characterized by prolonged stay, the object was located in this part of

the esophagus. Because of the proximity of the aorta, the danger of perforation of this vessel through ulceration is always imminent. Such an ulcerative process even though rapidly progressive may produce no symptoms until actual perforation has occurred.

An example of prolonged stay in this situation is the case reported by Bronner (4) of a boy five years who swallowed a coin. For twenty-two months there was a spasmodic cough, hoarseness and occasionally attack of pain re-

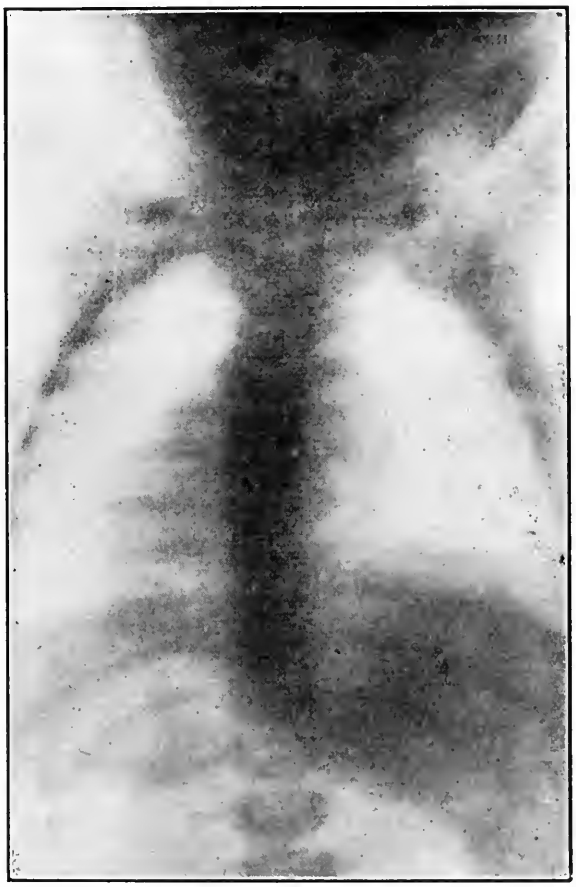

Fig. 4.—Case IV. Open pin in esophagus.

ferred to the abdomen. The swallowing of solids was difficult, fluids passed readily. Examination by means of the X-ray showed the coin in the esophagus opposite the fifth dorsal vertebra. It was pushed down into the stomach and discharged per rectum five days later. In Rectenwald's (5) case a coin was impacted in this portion of the esophagus for 129 days. In a case reported by Mac Intyre (6) and also in Kallionzis's (7) case, a coin was impacted in this situation for six months. In a case reported by Fullerton (8) a coin was impacted in this situation for seven months, giving no symptoms until three weeks before operation.

A case showing a still longer retention is that reported by Jalaguier (9) who removed a coin from the esophagus of a child four years old. It was swallowed when the child was sixteen months old, and for two years gave no appreciable symptoms. At the end of this time the child began to suffer from regurgitation, dribbling of saliva and vomiting. Examination with the X-ray showed the coin in the esophagus opposite the fourth dorsal vertebra. At the operation, the esophagus about the site of the impaction was found ulcerated and thinned. Halstead (10) reported the case of a child of five years who since infancy had suffered from vomiting, regurgitation and repeated attacks of abdominal pain. The diagnosis of congenital stricture of the esophagus had been made and with the object of finding the stricture an X-ray examination was made. This revealed the presence of a coin (cent) in the esophagus, impacted at a point about opposite the fourth dorsal vertebra (aortic constriction). It had been in this situation for at least four and one-half years.

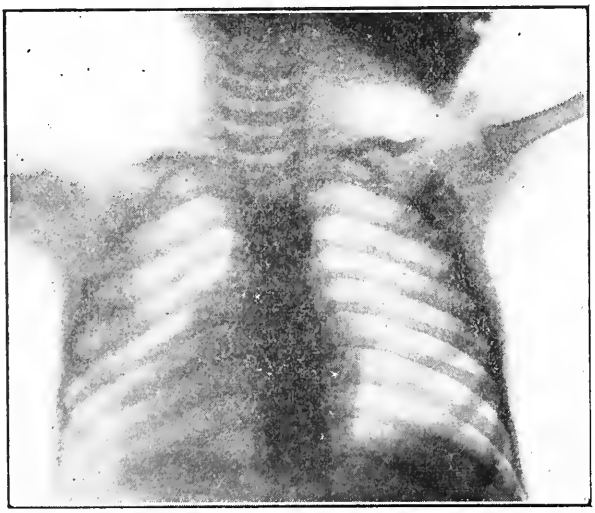

Fig. 5.—Case V. Disc whistle in esophagus, at point of aortic narrowing.

In the following case a disc whisttle was impacted in the aortic constriction of the esophagus.

Case V. Abe G., age 3 years, swallowed a tin whistle eight days before. For four days there were no symptoms. The stools were watched after the administration of a cathartic, but the whistle did not appear. Frightened at the non-appearance of the offending body, the parents brought the child to Dr. David Katz, who passed a soft rubber bougie into the child's stomach without any apparent hindrance or difficulty. Recognizing, however, that this did not rule out the presence of the body in the esophagus, the patient was referred for radiographic examination. The child was not seen, however, until three days later, during which time there had been a dry cough, dysphagia, and regurgitation of solid food. Fluoroscopy showed the disc whistle in the esophagus at a point about opposite the fourth dorsal vertebra, as is shown in the radiograph (Fig. 5). With the aid of a "coin catcher" the whistle was removed without any difficulty.

Case VI. Hannah W., aged 9 months, was referred to Dr. S. Newman with the history, that three days before the child swallowed a small safety-pin.

For many hours there was choking cough, dyspnea and nausea. Gradually these symptoms abated and the child seemed to suffer no discomfort. For the preceding twenty-four hours the child had again become very restless and refused nourishment. The head is held rigid and turned somewhat to the left side, and movement seems to cause pain. A radiograph (Fig. 6) showed the safety-pin open in the esophagus, the point to the left. Dr. Charles Elsberg performed an esophagotomy and found the point of the pin imbedded in the wall of the tube, and in order to remove it, it was necessary to divide the pin and extract it in two pieces. The recovery of the child was uneventful.

Impaction at the point of diaphragmatic constrictions is a rare occurrence, and foreign bodies are usually not well tolerated in this situation. Singultus is

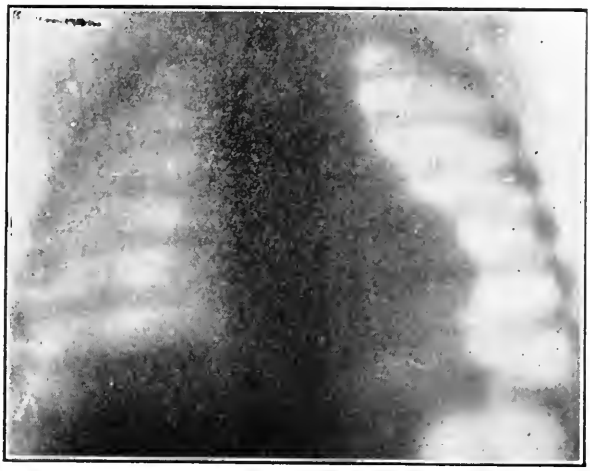

Fig. 6.—Case VI. Open safety-pin in cervical portion of esophagus.

frequently a persistent symptom from the beginning. There may be vomiting associated with a constant pain in the episternal notch or in the abdomen. Baldwin (11) reported a case of a girl who without apparent cause began to have symptoms of croup with dysphagia and regurgitation, which gradually increased in severity so that at the end of a month the child was considerably emaciated. A radiograph at this time showed a button in the esophagus five centimeters above the cardia.

The following case illustrates the usual history of bodies in this location.

Case VII.—Abe S., aged 8 years, was presented to me with the history that thirteen days before he had swallowed a disc whistle. At first he regurgitated all food, but later he was able to swallow fluids with but slight difficulty. He had singultus and complained of pain in the upper abdomen. General physical examination yielded no data having bearing upon his complaint. Radiography (Fig. 7) shows the presence of the whistle in the diaphragmatic portion of the esophagus. As a result of attempts at removal the whistle fell into the stomach and was passed later.

Tolerance.—If the primary symptoms persist and increase in severity, as happens but rarely, the difficulty in diagnosis is not great. As is most often the case, however, the primary symptoms either abate or subside completely, leaving the patient comfortable, or subject to but slight discomfort. During this period,

which may be of longer or shorter duration, the body thus lies in the esophagus without manifesting its presence. If the history of the ingestion is clear, the subsidence of the symptoms and the state of comparative comfort into which the patient passes lead to the belief that the body has entered the stomach and will in due time appear in the stools. If the history of the ingestion is doubtful, the return of the patient to an apparently normal condition will allay the suspicion of its entry, which the sudden and alarming primary symptoms may have aroused.

Though not disposed because of its structure to the retention of foreign objects, yet the tolerance that the esophagus exhibits to such substances as do

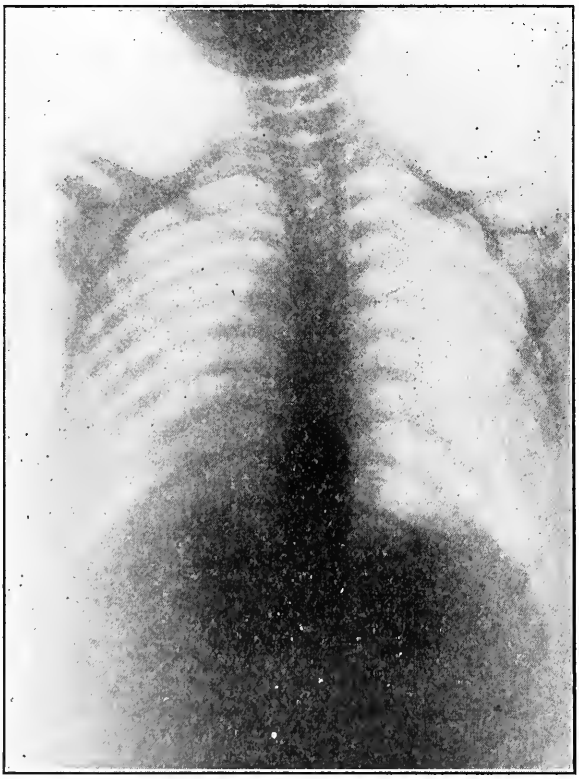

Fig. 7.—Case VII. Disc whistle in diaphragmatic constriction of the esophagus.

not cause much obstruction is remarkable and, as is to be noted from the cases cited, coins, buttons, pins, etc., have remained within the gullet for years before the onset of secondary symptoms. In many cases a sort of encysting process takes place, due to the swelling of the mucous membrane. Sooner or later, however, the gradual or sudden onset of the secondary symptoms of an obstructive or inflammatory type, serves to focus attention to a condition believed to have terminated.

Secondary Symptoms.—The secondary symptoms which appear depend upon the pathological condition, and upon the part of the esophagus affected. The edema which results from the pressure at the site of the impaction may go on to ulceration with the eventful perforation of the tube. An acute esophagitis or

periesophagitis, with or without abscess formation may be set up and the abscess may perforate either externally into the surrounding tissues, or into the interior of the canal. When the inflammatory process involves the cervical part there will be intense pain referred to the neck and chest, vomiting, dysphagia, chills and fever. The head is held rigid and every movement causes considerable pain. There is a dribbling of saliva from the mouth and frequently a severe cough. The entire region of the neck is swollen. If an abcess forms it may be recognized by the usual means. If the process occurs in the thoracic portion, perforations into the mediastinum, trachea, pleura or pericardium are possible. Rosenthal (12) reported a case in which a fragment of bone impacted in the esophagus of a boy had perforated into the pleura, causing pneumothorax. This and similar complications nearly always prove fatal. In another case ascarides were found to have perforated the esophagus, and to have entered the left pleural cavity, causing pneumothorax. In a case reported by Marshall (13) a stud-button impacted in the esophagus of a child seven months produced consolidation of the right lung, perforation into the trachea an inch above the bifurcation and pus in the bronchi and esophagus. Ulceration of the mucous membrane of the esophagus may take place insidiously without the production of marked symptoms. Hemorrhage resulting from this has in rare instances appeared as early as the eighth day.

In a case recorded by Heaton (14), blood was brought up within a few hours after the impaction of a disc whistle in the esophagus. In a case in which severe hemorrhages appeared on the eighth day, a deep ulcer was found eroding into the aorta. Hawley (15) reported a case of a boy of four years, who after three severe attacks of hematemesis died. At the autopsy a coin (half-penny) was found impacted in the esophagus about one inch below the level of the aortic arch. At one side where the coin came in contact with the esophagus was a deep ulcer; the one on the left side communicating with the descending sorta. The coin had been in this situation for six months. Rolleston and Whipman (16) reported a case of a girl of three years who after an illness of three months died, apparently from tuberculous peritonitis. The autopsy, however, showed a headless pin four and three-quarter inches long, resembling a hatpin, in the esophagus with two and one-half inches of its pointed end projecting upward and perforating the wall of the tube on the right side just above the level of the diaphragm. The other end had also perforated the esophageal wall, pierced the diaphragm and perforated the superior mesenteric artery, forming a false aneurism between pancreas and duodenum.

Fortunately, at the present time such sequelae or complications are not common, for our improved methods of diagnosis enable us to recognize the condition in its early stage and to remove the offending body before such dire effects ensue.

Foreign Bodies in the Stomach.

When, whether mechanically or naturally impelled, the foreign body enters the stomach, complete relief of the symptoms peculiar to esophageal impaction usually occurs. Frequently, however, the pain originally due to the presence of the object in the esophagus, persists for some time after the radiographic examination demonstrates the body in the stomach or intestines. So, also, the ulceration which may have resulted from its presence, may continue to give symptoms in spite of the irritating factor being removed, or an inflammatory condition aroused by attempts at removal may progress in spite of the success of such measures.

The actual entry of the foreign body into the stomach is rarely attended by any symptoms even though the object be sharp, nor are there any manifesta-

tions if it remain in the viscus but a short while. The symptoms depend upon the changes produced in the stomach wall and upon the alteration in the nature of the gastric secretion. Such is the tolerance exhibited by this organ, that these effects do not attain any degree of severity unless the object has been within its lumen for a long time. Sands reported the case of a girl who, during a fit of laughter, had swallowed a pen-knife with the blade open. For a short period after the accident there was severe vomiting and cramps, but for several days after this, until the removal of the foreign body by gastronomy there were no symptoms.

Once having entered the stomach, foreign substances usually may remain there but a short time, but may be retained days, months or even years. They

Fig. 8.—Case VIII. Hat-pin in the stomach and esophagus of a child. The circular shadow at the point of the pin is that of a blood clot.

may be vomited immediately upon entrance or not until vomiting has become a persistent symptom because of a resulting gastritis. The stomach does not show any particular tendency to impede the passage of foreign substances. When retention does occur it depends upon a multitude of factors, among which those due to the physical characteristics of the invading substance are the more important. It may be stated in a general way that the sharper, larger or longer, the object is the slower and more difficult will be its passage through the pylorus. Long bodies usually are found lying parallel to the long axis of the organ. When the object is very long it may be unable to completely enter the stomach, and it may then lie partially in the esophagus, as in the case, which through the courtesy and kindness of Dr. Frederick Gwyer, is here reported:

Case VIII.—A girl aged 20 months was admitted to Dr. Gwyer's service in Bellevue Hospital with the history that fifteen days before the child was noticed by its mother playing with various articles. That child's play was not interfered with because with the exception of some long hatpins there were no articles which it was thought could possibly harm her. After a while, on being lifted, it was noticed that she coughed a little. The possibility of the little girl having

swallowed something immediately suggested itself, but a glance reassured the mother, as she found her previous impressions correct, in that there seemingly was nothing there which the child could swallow. Soon after, however, one of the pins was missing and could not be found. It was then noticed that the child was awkward and held the body stiff when she stooped. Occasionally pain was complained of under such and similar circumstances. For the following three days the child had slight fever, cough, and complained more often of pain. Ten days after onset she passed a small quantity of blood in the stools. Twelve days after a radiograph, made by Dr. A. Holding (Fig. 8), showed a hatpin in the stomach and the esophagus, the oval head lying in the stomach and to the left, while the point was in the esophagus about opposite the fourth dorsal vertebra. There was no vomiting, the appetite was good, the bowels regular, and she slept

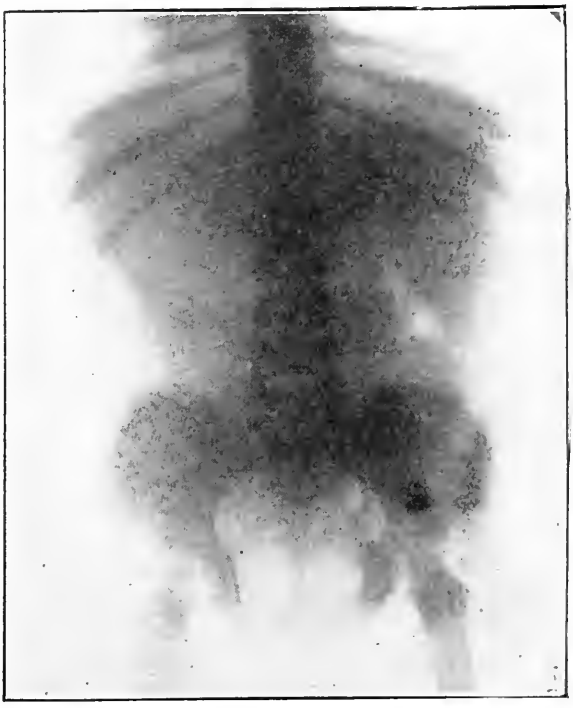

Fig. 9.—Case IX. Button in cecum.

well. On admission to the hospital the temperature was 98.6° F., pulse 106, respiration 24. Gastrotomy was performed by Dr. Gwyer a few hours after admission. The stomach was opened and the head of the pin found pressed against the posterior wall. It was drawn out with ease. The recovery was uneventful. The pin measured six and five-eighth inches in length and consisted of a steel shaft with an egg-shaped opalescent glass bead. This case is of exceptional interest not only because of the length of the object, but because of the few symptoms present and the absence of any marked damage to the esophagus or other structures.

A case in some respects similar to this was reported by Ballance. An infant of fifteen months swallowed a long hat-pin which was lodged in the lower

end of the esophagus, with its head at the level of the cardiac end of the stomach. The only symptom present was dysphagia. The pin was removed by gastrotomy.

A case of this kind was also reported by Hubbell (17) and is remarkable because of the age of the child, which was ten weeks. The patient presented no symptoms except pain and discomfort in certain portions of the body. Examination disclosed a prominence about one inch above and to the left of the umbilicus, and the operation resulted in the removal of a darning needle one and three-quarters inches long from the stomach.

In the majority of cases the entry and passage of the foreign body into and through the stomach is uneventful. There may be nausea, vomiting and pain. This is sharp, intermittent and cramp-like and is usually aggravated by the ingestion of food. The vomitus may contain blood. These symptoms, in some cases, appear promptly with the entry of the foreign body, and continue with more or less severity until its expulsion. In other cases the symptoms gradually abate and finally cease, as the stomach accommodates itself to the unusual condition. In the majority of cases, however, the distressing symptoms do not appear until a long time after the entry of the object; in other words, they constitute the secondary manifestations. It frequently happens that foreign substance which was long believed to have passed out, is ejected during the latter manifestations.

Bruch (18) reported a case of a child of eight months who swallowed a metallic object seven by two centimeters, and vomited it 69 days later. Adler reported a case of a child aged three and a half years who swallowed a copper coin. From time to time the child vomited and complained of pain in the abdomen. One hundred and two days later, during a very severe attack of this kind, the child fainted. Attempted restoration was followed by vomiting of the coin. Not only smooth but sharp bodies may be expelled in this manner. The symptoms distressing in their nature vary in their intensity at different periods in the same individual, remit for a while only to recur, and when persistent lead to marked emaciation.

Though usually single, in a few cases reported, quite a variety of foreign substances have been found in this organ. Perhaps the largest number present in the stomach of a child was extracted by Mayo Robson (19) through a gastrotomy incision, the patient being a girl of ten years. There were 42 cast iron garden nails one and five-eighth inches long, 93 brass and tin tacks, 12 large nails three collar studs, one safety-pin, one sewing needle. For a long time the only complaint was failure of the general health and loss of flesh. One day before coming under Robson's observation the vomiting of blood-stained material and severe abdominal pain appeared. The expulsion of a nail in the vomitus gave a clue to the diagnosis.

The objective examination of the children with whose stomachs there is resident a foreign substance, yields but a few data. In some cases palpation may reveal a tumefaction in the umbilical region which is tender to pressure and occasionally the foreign substance itself may be indistinctly felt. As a rule, freely movable tumors due to this cause are usually found in the lowest part of the stomach when the erect posture is assumed.

In the great majority of cases, whether its presence in the stomach has caused symptoms or not, the foreign body passes through the pylorus into the duodenum. The attempt on the part of the stomach to accomplish this is very energetic and persistent and unless a marked disproportion between the object and the pyloric orifice exists or because of certain characteristics of the former, as extraordinary length or sharpness, this termination is usual, though it may take place months or even years after its entry. As illustrating the active expulsive tendency on the part of the stomach is the experience of Markoe in the

case of a child of seven years who was presented with an indefinite history of swallowing a pencil six inches long. Gastrotomy 24 hours after the accident showed the stomach to be empty and the pencil lying in its entirety within the duodenum.

In its passage through the pylorus, the foreign substance may become impacted within this opening with dire sequelae. This accident is rare. A case has been reported, which Poulet cites, of a child who died of inanition after swallow-

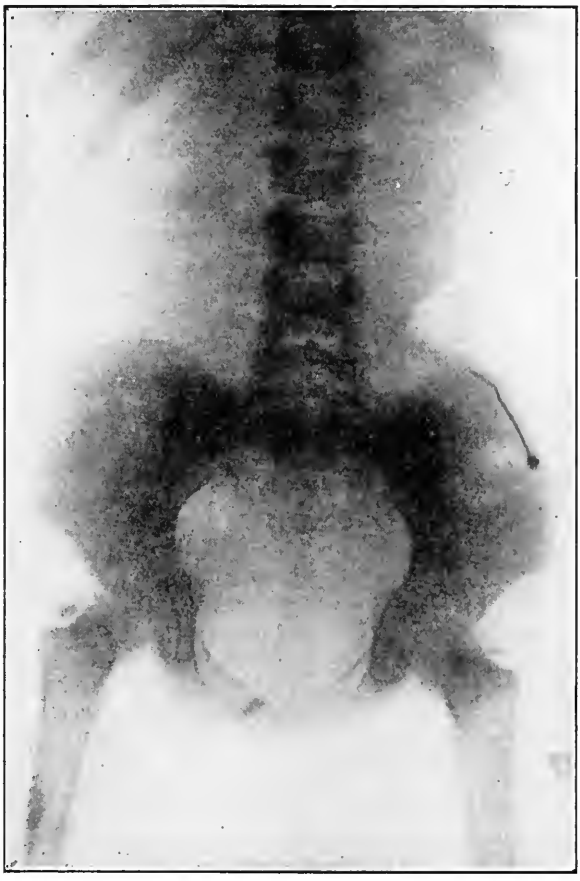

Fig. 10.—Case X. Scarf-pin in cecum.

ing a coin which at autopsy was found to be impacted transversely in the pyloric opening.

With sharp bodies there is also to be considered the possible though not frequent complication of perforation of the stomach wall. This is evidently an uncommon occurrence considering the frequency with which bodies like pins and needles transverse and even remain within its lumen without producing the complication. The stomach owes its immunity from this accident to the active movement of its thick wall and its power of reacting by rapid peristalsis to irritation of its mucous surface. The tendency to perforation is greater if for one reason or another the foreign substance is fixed and immobile and this

predisposes to the production of the complication even by a dull body. When this does occur the foreign substance may enter peritoneum, adjacent viscera or abdominal wall with general or local peritonitic inflammation.

Foreign Bodies in the Intestinal Tract.

The pyloric opening to a degree safeguards the intestine and prevents the entry into it of such substances as are likely to endanger its integrity or aff ct the general economy. Except the duodenum and at the ileo-cecal valve the small intestine throughout its length does not offer any particular difficulty to the passage of foreign bodies and is more tolerant to their presence and prolonged stay than is any other part of the alimentary tract. The majority of such objects as escape through the pylorus pass through the small intestine rapidly and without producing symptoms. It is unusual even for a large body moving through the intestine without arrest of its motion to cause even mild colicky pains. As a rule the presence of symptoms indicates arrested progress and their severity will depend upon the site of arrest, the nature of the body and the duration of its stay. The tortuosity and fixity of the duodenum, and the narrowness and conformation of the gut at the ileo-cecal junction, are anatomic conditions which predispose to the production of complications during the passage of an object through these parts.

Retention of elongated substances in the doudenum is not an uncommon complication. Lucas (20) reported the removal of a nail two inches long from the second part of the duodenum of a child who had swallowed it four weeks before. Radiographic examinations during the time showed it not to have changed its original position to the right of the spine, reaching from first to fourth lumbar vertebra.

Perforation of this part may take place soon after the entry of the object. It has been reported as occuring without symptoms. Bleisner (21) reported a case of an infant who swallowed a needle. The child died but not as the result of the accident. At autopsy the needle was found fixed in the wall of the duodenum which it had perforated at a point where the organ makes a turn over the pancreas. Such instances are, however, unusual, and as a rule the occurrence of this complication is usually attended by characteristic symptoms. In a case under the care of T. C. English (22) a girl of 16 swallowed a broken bodkin two and one-half inches long. There were no symptoms for five days and then persistent sharp pain referred to the right hypochondrium appeared, and there was tenderness to pressure of this region. A radiograph showed the object lying transversely to the right of the spinal column above the umbilicus—evidently in the duodenum. A second and third examination showed it to the left of the median line and below the umbilicus, and it was believed to have passed into the colon. The sticking pain in the hypochondrium persisted and 14 days after the accident it became very severe, radiating to the axilla. With this there was extreme tenderness to pressure and rigidity of the entire abdomen. At operation the bodkin was found to have perforated the duodenum.

Once having weathered this point the journey is usually rapid and uneventful until the cecal region is reached. All substances are arrested for a longer or shorter time at the ileo-cecal junction or within the cecum, and those of large size are capable of causing marked intestinal obstruction at this point. Rudis Jicinsky (23) reports two cases in which the symptoms of intestinal obstruction were produced by foreign bodies. One case was a boy of 10 years who swallowed a tin disc whistle which was found causing obstruction at the ileo-cecal valve and the other case, a boy of 12 years, the severe obstructive symptoms were found to be due to a wooden whistle impacted in the small intestine. Small

bodies may remtain in the cecum for a long time without symptoms, as in the following case.

Case IX.—Robert R., aged 2 years, swallowed a bone collar-button, which the radiographic examination (Fig. 9) twenty hours later showed to be lying in the cecum, where it remained for eight days, being passed on the eleventh day without having given any symptoms.

Rarely small bodies retained for a long time within the cecum may gather about themselves a sufficient incrustation of fecal material to cause obstruction.

Even sharp bodies proceeding point in advance are usually expelled and frequently a long time after ingestion, without causing symptoms or doing any appreciable damage to the intestine. Parks (24) reported the case of an infant of nine months who swallowed a scarf pin two and one-half inches long which was passed the following day without symptoms. Dupont (25) reported the case of a child 12 years who smallowed a pin which was expelled per anum in five days without any symptoms. Hall (26) reported a case of a child of five months who swallowed a safety-pin which a radiograph taken 36 hours later showed to be lying open in the stomach. Twenty-one months later the pin was passed per anum without symptoms. In Littig's (27) case a child of 11 months passed without any apparent symptoms a small safety-pin in a few hours less than five days and in Clemson's (28) case, an open pin of a similar sort was passed in 27 days without any symptoms by a child of six and a half months. Owens (29) and Solomons (30) reported cases in which similar foreign bodies were passed. Rodgers (31) reported a case of a child of 15 months who swallowed a glass-headed pin three inches long. It was passed in six weeks without symptoms. In a similar way the passage of sharp foreign objects in the following two cases was uneventful.

Case X.—A. C., age 12 years, was brought to the admission office of Bellevue Hospital with the history of having swallowed a pin ten hours before. General physical examination was negative. Radiograph examination (Fig. 10) showed the presence of a scarfpin in the cecum, lying with the head downward. It remained in this position for four days, but was finally passed six days after its ingestion.

Case XI.—B. M., aged 2½ years, swallowed a blunt screw about one and one-half inches long. Five hours after the accident fluoroscopic examination show it in the cecum (Fig. 11), where it remained for seven days, being finally passed on the ninth day after ingestion without any symptoms.

When present, the symptoms due to impaction of a foreign object in the intestinal canal may come on quite suddenly. Pain is an early, and usually persistent symptom, being colicky in character and referred to a particular part of the abdomen depending upon the site of impaction. It is increased by pressure, in this respect differing from colic due to simple spasm. In some cases it is the only symptom, but usually nausea and vomiting are also present. Though constipation is the rule, there may be diarrhea with blood in the stools. The persistence of the symptoms over a long period of time puts the patients in an extremely debilitated condition. Should the foreign body resume its march outward, the symptoms abate, only to recur if arrest of movement again takes place. With the passage of the substance into the rectum permanent relief is usually obtained. The duration of the stay of a foreign body in the alimentary tract is extremely variable, from days to months or even years.

Secondary Symptoms.—The presence or absence of secondary symptoms depends upon the nature of the foreign substance and its length of stay in the gut. On account of the tolerance exhibited by the intestine, the symptoms of intestinal obstruction or perforative peritonitis, may come on long after the

entry of the foreign substance, when the occurrence may have been forgotten. Persistent localized pain with a stationary condition of the foreign body as shown by x-ray examination demands operative interference in order to avoid impending disaster. Though there is greater liability to the production of a pathological condition by a sharp body than by a smooth one, even the latter may by continued pressure cause ulceration. This may progress with such severity as to cause a local peritonitis and also perforation with extrusion of the substance into the peritoneal cavity or adjacent viscera.

Because of constant localized pain in the right iliac fossa, Moullin (32) operated upon a girl, and found a broken hat-pin, over four inches long, in the small intestine just above the ileo-cecal valve. The pin had been in the alimentary tract five days.

The symptoms due to perforation may be the first and only symptoms re-

Fig. 11.—Case XI. Screw in cecum.

resulting from the ingestion of a foreign object. Their appearance after a long period of quiescence may result in their being ascribed to another cause. Menracher (33) reported the case of a girl of six years who swallowed a hair-pin. For four weeks after this a constipating diet was administered and observation of the stools maintained, but the pin did not appear. As the child suffered no inconvenience, the precautions were relaxed at the end of this time. During the four months following there was little evidence of any trouble. At the end of this time such acute abdominal symptoms developed as suggested the diagnosis of appendicitis or intestinal perforation. Preparations for operation were made, but in a few hours such remarkable improvement in the patient's condition occurred that the operation was deferred. Twelve hours later, however,

the symptoms returned in full violence and then an abdominal incision disclosed a diffuse suppurative peritonitis, which was found to be due to perforation of the duodenum at the junction of descending and ascending parts, by the blunt end of the hair-pin.

The role of the appendix in restraining foreign bodies is overestimated. Quite a variety of such substances were found by Mitchell at autopsy, within the appendix—grape seeds, fragments of bone, shot, nutshell, etc., without any inflammatory stigmata being present. In children these findings are exceedingly rare. The presence of such a body within the appendix may cause appendicular colic due to the attempted expulsion of the intruding substance. This may occur without any accompanying inflammation. As a result, however, of laceration or ulceration of the mucous membrane an inflammatory process may be initiated which may progress to suppuration and perforation. Glazebrook (34) reported the case of a child of 14 months who suddenly became ill with a severe chill and rapid breathing and cried with pain on being moved, the pain apparently being referred to the lower right chest. The child's condition rapidly became worse, she lapsed into coma and died three and a half hours after the onset of the illness with what were considered to be pneumonic symptoms. The autopsy showed a purulent inflammation of the appendix with localized peritonitis, due to perforation of the middle part of the appendix by a pin which lay partly within its lumen, the porcelain head lying about three-eighths of an inch above tip. The pin had been in the alimentary tract for over 10 months and had never given symptoms until the fatal complication took place. Kellock (35), operating on a child for inguinal hernia, found the appendix in the hernial sac and a pin within the appendix.

Foreign Bodies in the Rectum.

With the entry finally of the foreign object in the rectum after an uneventful or eventful passage, the liability to the production of complications is at a minimum. This part of the gut does not tolerate the presence of foreign substances and in the great majority of instances, they are rapidly expelled and usually without symptoms. If the body be large or sharp, the persistent efforts at expulsion may cause pain, tenesmus and dysuria. Constipation is usually present and the small movements may be accompanied by a mucous discharge. Blood may appear in the stools either as a result of laceration or ulceration. In many cases, the foreign body may give rise to symptoms only at the moment of attempted expulsion, as in a case of my own cited further on. Great is the variety of substances thus manually extracted because of local symptoms, from the bust of Napoleon to toy automobiles, etc.

Foreign bodies may cause obstruction by acting as valves at the anal orifices, as in the case reported by Mackay (36). A child of two years, suffering from debility and malnutrition, was presented with a history of obstinate constipation for three months. Examination showed an enormously distended abdomen so tympanitic to percussion that liver dullness was obliterated. Rectal examination was impossible until dilatation with forceps was done, and then a button was removed which, acting like a valve, had closed the anal opening and caused the obstruction.

Perforation of the wall may take place. The fixity of this part of the gut, the prominence of its valvulae and the consistency of the feces all predisposing to this occurrence. It is in the lower four inches that this takes place more frequently than the upper. As a result of either ulceration or perforation abscess formation may occur, involving the cellular tissue about the rectum.

Foreign Bodies in the Respiratory Tract.

The inhalation of a foreign body in the respiratory tract is attended by symptoms usually not differing in their nature from those due to the entry of such body into the alimentary tract. Indeed, the similarity of the initial symptoms is so close that it is impossible to tell from the consideration of these phenomena alone which tract has been invaded. Such a fatal termination as rarely occurs from prolonged spasm of the glottis excited by impaction of a large body, high in the esophagus, may also occur with similar suddenness from the presence of a foreign substance in the upper larynx. In the same manner, also, as it often enters the esophagus without any symptoms, so a foreign body may pass through the larynx and trachea into the bronchi with symptoms exceedingly mild and transitory.

In this accident, as the object is carried to the back part of the mouth in the act of swallowing, a sudden inspiration, voluntary or involuntary, sucks it past the open glottis into the lower larynx, trachea or bronchi or impacts it into the upper larynx, more or less mechanically obstructing the glottic opening. This sudden inspiration may be the result of the expression of such emotions as fright or joy or a sudden coughing paroxysm. The attempt at removal of a foreign body from the pharynx or esophagus sometimes results in its being introduced into the air passages or material ejected during vomiting may be sucked in by the inspiratory movement which follows this act. Of the foreign bodies entering the larynx those which by virtue of their size are small enough to pass through the sensitive glottis, initiate in their passage reflex irregular contractions of the laryngeal and respiratory muscles, causing severe dyspnoea, cyanosis, suffocation, slow pulse and collapse, a syndrome termed glottic spasm or the initial paroxysm. It has been found by Crile (37) in experiments on animals that the pronounced respiratory and circulatory disturbances result from irritation only of the upper and middle part of the larynx; irritation of the mucous membrane of the lower larynx or of the trachea not producing such reflex effects. Rosenthal has shown experimentally that irritation of the superior laryngeal nerve causes inhibition of inspiration with strong expiratory movements, at the same time that the larynx is raised and peristaltic contractions of the esophagus take place, the mechanism having for its object the expulsion of foreign body which may reach trachea or larynx. The severity of the spasm depending as it does upon the degree of irritation accounts for the mildness of this phenomenon in some cases. When the body is small or smooth there may be no spasm. As a result of the intense expiratory efforts the glottis may be forced open and the foreign body may be expelled immediately after its entry with complete relief of the symptoms. If the cough and expiratory efforts are futile, then the inspiratory air current tends to draw the offending body deeper into the larynx, trachea or bronchi, its final resting place depending upon its shape and size. It may lie partly in the trachea and partly either in right or left bronchus. This is the usual position of long bodies, while small round bodies pass to such a bronchus as corresponds to their largest diameter. The right bronchus is more frequently invaded than the left, both because of physical and functional peculiarities, the former tube having a large diameter (its relation to the left being as 100 to 77.5), the intrabronchial septum being situated to the left of the median line, and the aspiratory power of the right lung being greater than the left. The narrowest part of the lumen of the trachea is at its commencement. From this point it gradually grows wider until the middle of the tube and then grows smaller until its bifurcation. The bronchi exhibit a similar variability of their lumen.

Irregular bodies may rest in the lower larynx or trachea and when not imbedded or too large move freely in the air current. This mobility obtains also during the early period of the sojourn of small bodies and they may by irritation of the sensitive mucous membrane of the upper larynx cause single or multiple repetitions of the symptoms characterizing the initial paroxysm. It is obvious that obstruction of the larynx or trachea is far more dangerous than that of any other part of the respiratory tract, because the respiratory tract at this point is a single tube.

Frequently during the paroxysm of coughing the body may be thrown from

Fig. 12.—Scarf-pin in cecum.

one part of the bronchial system to another, into the trachea or larynx. In a case reported by Naismith (38) a boy of seven years inspired a glass bead which rolled about the trachea, then obstructed the right and then the left bronchus and was eventually coughed up. Brown (39) reported a case in which a bullet in the bronchus of a boy of seven years was displaced from the left to the right side by coughing. In a case reported by Morris (40) a large pin was cougned from one of the main descending bronchi into a minor ascending branch.

It may be expelled from the respiratory tract, swallowed and enter the esophagus. According to Halstead this occurs frequently without the fact ever being known. Warren wrote of an infant who expelled a horse-nail from the bronchus, swallowed it and passed it later in the stool.

The stormy period is eventually followed by a state of comparative comfort when the body becomes fixed. Relief is, however, not thus wholly obtained from

discomfort, for with impaction and the swelling of the mucous membrane comes a new train of sequela. If the foreign body is such as is capable of swelling in the presence of fluids, the respite is short, and the secondary symptoms appear as soon as the swelling of the foreign body obstructs the lumen of the tube within which it lies.

After a longer or shorter interval, suppuration about the body may again render it mobile and liable to extrusion from the organism with the products of suppuration. Wassinger reported a case of a boy of 14 years who inspired a brass headed tack into his left bronchus, which was coughed up 18 months later with considerable pus. In a case reported by Rose (41) a girl of six years inspired a beech-mast (fruit of a beech tree) which was coughed up 10 years later with a pint of pus.

Tillman (42) reported a case of a girl four and one-half years who aspirated a cherry pit into the stomach. This was followed by pulmonary gangrene and empyema. Thoracotomy was done for complications but the foreign body was coughed up two years later. The course of events were similar in a case reported by Matthew of a girl of five years who aspirated a tack into the left bronchus. The tack was coughed up long after the operation for complications had been done.

This termination by expulsion may occur at any period but it takes place most frequently after the manifestations of secondary symptoms. Whether primary or secondary, expulsion occurs in about 60 per cent. of the cases. Bodies may remain in the air passages for intervals varying from days to years, without producing pathological changes of any significance, and be eventually coughed up. The longest stay recorded is 60 years; the foreign body being a bone, which was impacted in the right bronchus. (Gross.)

Foreign Bodies in the Larynx.

Foreign bodies which become lodged in the larynx, lie most frequently in its upper, supraglottic part. Impaction here is fraught with great danger, not so much because of the obstruction the body may offer to the passage of air into the lungs, but more so because irritation of the mucous membrane of this zone may result in inhibition of respiration and circulation with sudden death—the laryngeal spasm already described.

When lying in the ventricles of the larynx and neither causing marked injury nor obstruction, foreign bodies give but few symptoms. Cases have been reported in which pins, coins and other substances have remained in the pyriform sinuses for years without giving symptoms. Patterson (43) removed an open safety-pin from the larynx of a child of nine months. Advice was sought because of a fullness of the right side of the neck—there being no other symptoms. Direct laryngoscopy showed the pin lying with its point in the larynx, the protector in the right pyriform sinus and the hinge close to the right border of the epiglottis. It had been in this situation for six weeks.

Rosenheim (44) removed a button-hook from the larynx of a child seven years, but until two months before removal there were practically no symptoms except a slight aphonia. During the last two months the child had several attacks of laryngeal.

When in this position but so placed as to irritate the vocal chords or mucous membrane, it usually gives rise to a train of persistent distressing symptoms. Wherever it lies in the upper part of the larynx, the ever-present possibility of the production of serious complications exists; for a slight displacement may result in impacting the substance within the glottic opening or in initiating a spasm of the glottis. Aphonia, a persistent cough, with mild or severe attacks of laryngeal spasm, characterize the presence of a foreign body in this sensitive

area of the larynx. When the spasm occurs after an interval of quiescence and during an attack of coughing, it means that the body has been displaced into this region from trachea or bronchi.

With the subsidence of the alarming symptoms of the initial paroxysm and the impaction of the body in the larynx, the relief from distress may be complete for a long time; the duration of this period of tolerance depending upon the location of the body. In most cases, however, the voice soon becomes changed in timber or intensity, respiration is hurried, labored and irregular, and a persistent paroxysmal cough is present. Such patients are extremely restless, complain of pain in the throat which is made worse by phonation and deglutition. The coughing paroxysms are frequently followed by vomiting.

In impaction of an object within the lower larynx (that portion included within the cricoid cartilage) the symptoms will depend upon the degree of obstruction to the air current. If the interference with the entrance of air is not great, the foreign body will be tolerated and particularly if fixed, give but few symptoms. In the following case a tack which rested for four days in the lower larynx was not tolerated but gave severe symptoms because its point probably lay in the rima.

Case XII.—Fanny R., aged 12. Ten hours before coming under observation, while holding a tack within her mouth, she laughed. She immediately began to choke and gasp for breath, and then a few seconds later the dyspnea was relieved and she was comfortable. She soon, however, became very hoarse, phonation was painful and deglutition was difficult. She began to have a dry hoarse cough which caused severe pain. The child was in an extremely nervous condition and laryngoscopic examination was impossible, while the suggestion of narcosis for the purpose of examination was not accepted. A radiograph (Fig. 13) which was made after considerable difficulty, shows the tack in the lower larynx. Operative measures which were suggested were refused. After the first three days the symptoms gradually abated in severity, although from time to time there were severe paroxysms of cough with mucous expectoration. Five days after occurrence of the accident and during one of these attacks she expelled the tack. The cough persisted for several days, but the recovery was eventually complete.

A similar case was reported by Zia Noury (45) of a girl six years old who, while eating, had a sudden attack of laryngeal spasm with dyspnea, which lasted for a short time. She then began to suffer from hoarseness and pain in the neck during swallowing. Dyspnea gradually became severe. Thyrotomy showed small pieces of egg-shell in the larynx.

The case reported by Garel (46) is interesting in this connection. A girl aged two and a half who, while swallowing some salt, had an attack of suffocation. From that time on she suffered from aphonia and severe attacks of glottic spasm. The radiographic examination showed at the level of the larynx, a dark, vertical shadow as of some metallic substance. By means of Killian's tube speculum, a black mass was seen between the cords, which upon removal proved to be the copper hook of a hook and eye. The foreign body had been in the larynx for five months.

Secondary Symptoms.—The larynx does not, generally speaking, tolerate foreign bodies, and it is usually not long before inflammatory symptoms appear, either after partial or complete subsidence of the primary symptoms. Edema of the glottis may come on suddenly and threaten life. Usually, however, the onset of the symptoms is gradual. An acute laryngitis, which progresses to a chronic condition with ulceration and sometimes abcess formation, is the frequent march of events. The paroxysmal cough, the hoarsness, the dyspnea and

vomiting of this condition have caused it to be mistaken for asthma or tuberculosis, as in the two cases cited by Shirly.

Foreign Bodies in the Trachea.

Just as a foreign body originally lying in the bronchi may become secondarily impacted in the larynx, so may a substance, primarily impacted in the larynx, become displaced and fall into the trachea. If the body be of such size as to markedly obstruct the lumen of the tube, its presence is not compatible with life, simply on account of the asphyxia resulting from mechanical obstruction. Such large bodies seldom, however, reach the child's trachea, though a vegetable substance like a bean may, by absorption of secretions, swell sufficiently to completely obstruct the lumen of the tube. The great majority of such substances which become resident in the trachea, are small and the significant feature of this is, that they give but few obstructive symptoms. Irritation of the mucous membrane of this tube does not result in any pronounced reflex effects, consequently the presence of a foreign substance in this situation may arouse no symptoms but cough, while the examination of the lungs yields negative data. Jackson (47) reported the removal of a safety-pin from the trachea of a child one year old. The pin lay in this situation for one month, producing no pulmonary symptoms and the impression had been that it was lodged in the esophagus, until the Roentgen ray examination revealed its true location.

Secondary Symptoms.—The trachea tolerates in a remarkable way a foreign body which does not injure its wall or interfere with its patency. As a result of prolonged stay, a tracheitis, as evidenced by cough and frothy expectoration, may result and this inflammation may extend to the bronchi. This takes place earlier in those cases in which the foreign body is not firmly fixed but moves with the air current.

Foreign Bodies in the Bronchi.

The symptoms produced by foreign bodies in the bronchi depend both on the degree of interference with the functionating power of the lung and on the severity of irritation of the bronchial wall. Marked occlusion of one of the main bronchi is not compatible with life. Death does not come on suddenly or rapidly but the compensating power of the unaffected lung does not seem to be sufficient to enable the function of aeration to be satisfactorily performed. Obstruction of one of the minor bronchi does not produce such disastrous consequences. This is due to a compensating hypertrophy of the lung. In animals the tying off of one main bronchus results in death, from rupture of the other lung and pneumothorax or from stasis of blood in the pulmonary vessels.

In the vast majority of cases after the subsidence of the suffocation paroxysm incidental to the entry of the foreign body, and its impaction within the lumen of one of the secondary bronchi, there comes a period of calm.

The intruding substance seems to be tolerated and no manifestation of pathological changes are evident. Thus such foreign bodies as nails (Colard), coins (Mitchel, Baldwin, Dupuytren), pins, pieces of bone have been found in trachea and bronchi where they had lain for years without giving symptoms.

Bunch and Lake (48) cited 31 cases in which the foreign body was in the trachea or bronchus for intervals from one year to sixty, in all which recovery took place after expulsion or removal.

Symptoms.—The symptoms may be divided into those due to irritation of the lining of membrane of the bronchi, and those resulting from interference with the functionating power of the lung. Not in all cases are the manifestations similar. A slight cough, as the only symptoms in one case, will contrast with

severe dyspnea, paroxysmal cough with bloody expectoration in another. Cough is the most constant, severe and persistent symptom. In some cases the paroxysms are frequent and exhausting, and are accompanied by considerable mucous expectoration. Where the foreign substance has been in the bronchus for a long time, large quantities of frothy blood-tinged fluid may be brought up. This is usually swallowed and then expelled during the vomiting that often follows the severe coughing. In no disease except tuberculosis or pertussis is vomiting and coughing so constantly associated. Hemorrhages may occur. In a case which came under Huber's (49) observation repeated hemorrhages ceased only when an iron nail was coughed up after being in the bronchus for two years. Hoarseness may be an early and persistent symptom. Pain is not common, but when present it is referred in a general way to the chest or upper abdomen.

The respiratory rate is usually increased and either the inspiratory or expiratory phase, frequently the former, may be labored and difficult. The accelerated respiration appears to be rather constantly present even when no other symptoms are evident.

The children are at first very restless, but soon assume a position in which respiration may be carried on most effectively. Huber (50) has called attention to an important test regarding the position of the foreign body. He observed in cases in which the object evidently was not impacted, that the position of the patient would influence the symptoms in a striking and characteristic manner. Distressing coughing paroxysms resulted when the patients were disturbed or placed on the unaffected side; relief was immediate when the original posture was resumed. It appears as though such children instinctively assume a position in which there is least disturbance of the foreign body. When the passage of air into the lung is markedly interfered with, as in obstruction of a large bronchus, there are all the evidences of dyspnea, as the stooping position of the body, the contraction of the auxiliary muscles, the dilation of the alae nasi, etc. All the accessory muscles may be brought into activity. Cyanosis will depend on the degree of obstruction to the entrance of air. Hoarseness may be present. Fever is, as a rule, not present until the sequelae appear. In those cases where the obstructon is not marked the general condition of the child may in no way deviate from the normal.

Physical Examination.—The physical examination of the chest yields data which aid considerably in the establishment of the diagnosis. The signs, however, depend upon the amount of obstruction the foreign body offers to the entrance of air to the lung parenchyma. This does not depend entirely upon the size of the impacted body, for a small rounded body may completely fill the lumen of the tube, while another but larger body, either because of its irregular shape, or because it is perforated, like a whistle or a bead, or a tracheotomy tube may allow the entrance of considerable air.

If the bronchus obstructed be a minor one, examination of the chest may yield no positive data, because the signs which a small area of collapsed lung would give are over-shadowed by compensatory dilatation of the adjacent lung, a process which always takes place in greater or lesser degree.

When obstruction of one of the larger bronchi occurs, depending upon the site and degree of obstruction, there will be dullness, diminution of tactile fremitus, with diminution or absence of vesicular murmur and limitation of chest movement. The compensatory dilatation about the collapsed area of lung tissue obliterates the signs obtainable by percussion to a greater degree than

those obtainable by ausculation, so that normal resonance with absence of vesicular murmur associated with other essential data, is strongly suggestive of the presence of a foreign body in one of the bronchi.

In addition to the diminution of its intensity, the pitch of the respiratory murmur may be so raised as to acquire a sibilant quality. This is due to the passage of the air current through the obstructed bronchus and though in some cases it is not present at first, it becomes evident later as a result of further narrowing by the swelling of the mucous membrane at site of the impaction. The respiratory murmur may be simply coarse and noisy. In one case where the foreign body was a reed whistle, the whistling sound of this instrument was heard. In a girl who aspirated such a whistle into her right bronchus, Angleis states that the whistling sound produced by the rushing of the air current could be heard at a distance of 15 meters from the chest. As soon as inflammation of

Fig. 13.—Case XII. Tack in larynx.

the mucous mebrane is set up, loud subcrepitant rales will be heard over a localized area.

As has been mentioned when the foreign body is such as is capable of swelling in the presence of fluids, the symptoms following the initial paroxysm are exceedingly severe, persistent and grow progressively worse. This is due not only to the marked obstruction caused, but also because of the pressure exerted upon the bronchial wall. The suffocative paroxysms come on suddenly. In Kellock's case the initial paroxysm lasted 20 minutes after the inspiration of a bean. After being without symptoms for about 15 hours, the child began to suffer from severe pains in the chest; there was lividity, cyanosis, dyspnea and severe coughing. Examinations of the chest showed signs of deficient ex-

pansion, impaired resonance, feeble respiratory murmur and moist rales over the entire left lung indicating obstruction of the left main bronchus. The foreign body was removed through a low tracheotomy wound. Compared (54) reported the case of a boy seven years who aspirated a piece of husk. Intense suffocation and severe incessant cough followed, then fever, chills and pains in left chest. The cough grew worse and was accompanied by, first, sanguineous, then purulent and finally fetid expectoration. The diagnosis of large abscess at left base was made. The x-ray examination failed to show the foreign body. Bronchoscopy was done with difficulty because of enormous quantities of fetid pus

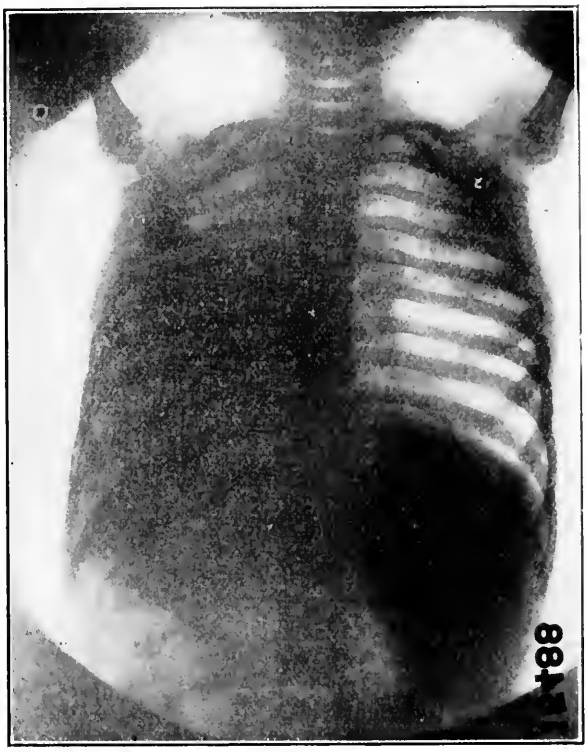

Fig. 14.—Case XIII. Nail in left bronchus.

but during the examination the husk was found obstructing the lumen of the examining instrument. However, in some instances the severe symptoms due to foreign bodies of this nature after reaching a climax may gradually subside, either because of disintegration or absorption. Thus in a case reported by Clayton (55) a boy of 12 years suffered severely from chills, fever and cough with hemoptysis, because of obstruction of one of the main bronchi in the right lung by a peanut. There was gradual recovery, however, due to disintegration of the foreign substance and expulsion of the fragments by coughing.

In concluding the consideration of the primary symptoms and signs it cannot be too strongly emphasized that in many cases they are singularly mild and few, particularly in the early history of the condition. The exception is in the class of cases just narrated. Frequently even though the early period be stormy, the symptoms gradually abate so that a state of comparative comfort

is soon reached, which continues to exist for a long time. Even with a portion of the lung atelectatic the only symptoms may be a mild cough. The confusion and difficulty in the establishment of the correct diagnosis that this may cause has already been dwelt upon.

Tolerance.—The bronchi tolerate the presence of a foreign body under conditions already stated. The extent of this period of tolerance, as well as the intensity and severity of the pathological process which succeeds it, depends to a greater extent upon the nature of the impacted substance and its location than upon the resistance of the organism. Septic organic objects will produce an inflammatory process more rapidly than an aseptic inorganic one. A non-septic

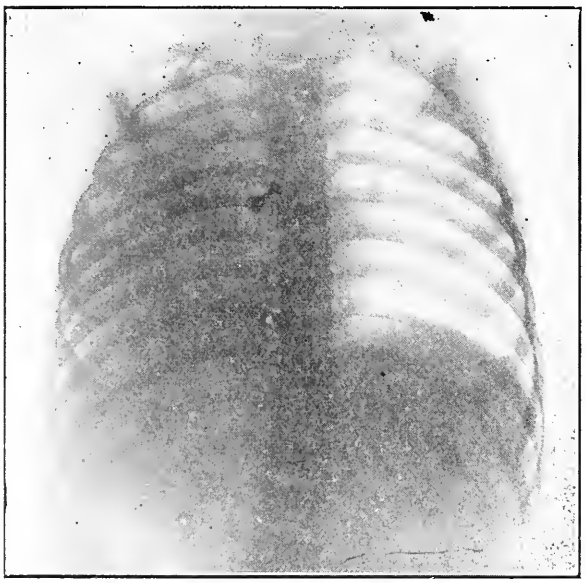

Fig. 15.—Case XIV. Collar-button in left bronchus

foreign body may lie within the lung for an indefinite period and produce no inflammatory changes.

When the inciting cause for the inflammatory process is an infective agent introduced with the foreign substance, a bronchopneumonia inflammation appears early, in some cases at the end of 24 hours. Otherwise the ulceration which later results from the pressure of the impacted body is the starting point of the inflammatory process. As this ulceration depends upon the position of the intruding substance within the bronchus, it is apparent that in those cases where its position is such that no pressure is brought to bear upon the mucous membrane, and no interference with its circulation takes place, the pathological lung changes may be delayed for an indefinite period.

Pathology.—Following the occlusion of a bronchus by a foreign body, compensatory dilatation of not only the adjacent but also the distant lung parenchyma occurs to a greater or less degree with the production of vicarious emphysema. This is due not only to the yielding of the alveoli but also to the yielding of the bronchioles and smaller bronchi.

If the obstruction is incomplete, during inspiration the air passes through the narrowed part and distends the bronchi and fills the air spaces, but the expulsion

of air is prevented by the plugging of the tube by inflammatory products, which act as ball-valves and thus the expiratory effort is spent in stretching the walls of the tubes. The inflammatory process set up by the foreign body further weakens the wall of the bronchus (as Stokes has pointed out, the inflammation of the mucous membrane paralyzes the subjacent muscular layers so that the tonicity of the elastic muscular fibers is lost), and it thus offers no resistance to the increased intrabronchial pressure resulting from the expiratory efforts and from the coughing spasms associated with the condition. When the obstruction of the bronchus is complete, collapse of the portion of the lung, which it supplies, occurs. The circulation of the blood in this atelectatic lobule is, however, still maintained. and when by direct extension the inflammatory process initated at the site of impaction reaches this atelectatic area the secretions accumulate and bronchiectasis finally results. Lichtheim has shown, experimentally, that this bronchiectasis is a direct result of the inflammatory process within a partially

Fig. 16.—Case XV. Blunt screw in bronchus of right lower lobe.

atelectatic lung. If no inflammation results there is no such dilatation and the air is absorbed. In the animal experiment of Lichtheim absorption was complete in 24 hours.

At first localized the process soon extends to the air spaces surrounding the affected bronchus, producing all the essential pathological changes of a bronchopneumonia, namely, the inflammation of the mucous membrane, the consolidation of areas of lung tissue, the diffuse bronchiectasis, the plastic or exudative pleurisy and the areas of atelectasis, all being reproduced. This pneumonia does not usually resolve but goes as an interstitial process with or without abscess formation. According to Tuffier (51) abscess occurs in 11 per cent. of the cases. It occurred in 29 per cent. of Wood's 89 cases. The abscess cavity may be in communication with a bronchus from the beginning, or it may affect a connection by perforation and the impacted substance loosened by purulent inflammation may be expelled during coughing.

There is a peculiar liability to gangrenous degeneration of the inflammatory products of a secondary bronchopneumonia due to foreign bodies. The gangrenous process eventually may extend to a lung previously unaffected by inflammation. In this gangrene, in addition to gangrenous odor of breath, and fetid expectoration which contains necrotic lung tissue, there may be another characteristic symptom—hemorrhage. This, according to Rilliet and Barthez, is present in about one-quarter of the cases and may be fatal. The inflammatory condition of the pleura may result in empyema or this may be brought about by the perforation of a lung abscess, with the extrusion of the foreign substance into the pleura. A case has been reported where, during a post-mortem examination, a foreign body was found in an empyema 13 years after aspiration.

The perforative process may cause pneumothorax. This condition has, however, been reported as having occurred early, due to the strain upon the unaffected lung by violent coughing efforts resulting from the impaction of a foreign body. This complication is rare. As a rule it is first abscess formation, then rupture of abscess, with pyopneumothorax. Ast (56) reported a case of a girl four years old who aspirated a pebble. There was an adhesive pleurisy with severe bronchopneumonia, and on the eleventh day perforation with pneumothorax.

Secondary Symptoms.—The secondary symptoms are to be considered as being those due to sequelae or complication of the presence of a foreign body. The striking features, whatever the symptom-complex presented, are the paroxysmal cough followed by vomiting, the evidence of chronic dyspnea, the extreme chronicity of the condition, associated with frequent attacks of acute pneumonic inflammations.

Restitution.—After the removal of a foreign body the signs within the chest clear up with remarkable rapidity. This has been commented upon by many observers.

Not only is this true of the early lesions but even in cases of long duration, the well marked pathological changes recede rapidly and the restitution of the lung to normal condition is almost complete. Thus such remote changes as the curvature of the spine, clubbing of fingers, all disappear. The cicatrization of productive inflammatory tissue does not to any extent impair the functional power of the lung. These changes are perhaps apparent only by Roentgen ray examination.

As serving to illustrate the difficulties which arise in the diagnosis of foreign substances in different parts of the bronchial system, the following cases are cited:

1. There are a class of cases in which the history of entry may be entirely absent. The etiological factor is only accidentally discovered in the study of a secondary condition which is not considered to have any relation to the presence of a foreign body.

Case XIII. Vincent M., age 5 years, was admitted to the Children's Service of Bellvue Hospital in the service of Dr. Howland, with the following history:

One and one-half years before, the child became ill with pneumonia, and was ill for sixteen weeks. The cough, which had been severe during this illness, then ceased for two weeks. From that time on, however, he has had more or less constant paroxysmal cough which is very frequently followed by vomiting. He is feverish towards evening. There have been no night sweats.

Physical examination showed a normally built child, breathing rapidly, but without any apparent dyspnea. The respiratory movements on the left side were somewhat restricted. The right lung was clear of signs. On the left side there was a marked dullness, both anterior and posterior, the note becoming flat at

base. The vocal fremitus was increased. Except for a small area above the second rib in front and fourth rib behind the breathing was everywhere bronchial with a few crepitant and subcrepitant rales. P. 120, resp. 36.

The blood showed 18,000 leucocytes, 78 polynuclear, 14 large mono and 8 small mononuclear.

The diagnosis of a probable abscess of the lung was made. The radiograph (Fig. 14) showed the presence of a nail in one of the bronchi in the lower lobe of the left lung. The entire lung, except for a small area at the apex, was densely infiltrated. The heart was not displaced. The other lung showed evidence of compensatory emphysema. With these findings in evidence the parents were questioned as to the history of the ingestion of such a substance. They then recollected that about two years previously the child had swallowed a nail and that a physician had been consulted, but since the accident was followed by no discomfort, nothing was done. When several months later the pneumonia appeared, the history of the ingestion of the foreign body was not considered to have any bearing upon the condition. Operative interference was refused and the child left the hospital. There were several attacks of pneumonia, and four years after its inspiration the child died with the foreign body not removed.

Case XIV.—S. P., age 5 years, was admitted to the Children's Service of Dr. LeFetra in Bellevue Hospital with the history of having been ill for five weeks with cough and fever, the latter symptom being present mostly at night. The coughing was severe and frequently followed by vomiting.

Physical examination showed a well developed and well nourished child. There was fullness of the left side of the chest with some diminution of expansion. There was dullness over the left chest posteriorly and in the axilla from the spine of the scapula to base. The breathing was of the bronchial type, though diminished and distant. Fine crackling and coarse rales were present. The heart was not displaced. The chest was tapped, but no fluid was obtained The clinical diagnosis rested between an encysted empyema and an abscess of the lung.

The radiographic examination (Fig. 15) disclosed the presence of a collar button in the left bronchus just beyond the bifurcation. The central portion of the middle and lower lobes showed considerable infiltration.

The foreign body was removed by Dr. S. Yankauer by bronchoscopy.

Case XV.—Max W., 5½ yrs., gave a history of paroxysmal cough followed by vomiting for three years. This had been diagnosed as whooping cough and asthma, and the child was treated for these diseases. Six months after onset of coughing he developed pneumonia and was ill twenty-one days. Two weeks later had another pneumonia. On the eighth day of the illness the child was sent to a hospital where the diagnosis of pleural effusion was made, but thoracentesis resulted negatively. The child improved slowly and left the hospital with a severe cough accompanied by bloody sputum and occasionally followed by vomiting. He lost flesh and strength rapidly. Several weeks later he was again admitted to the hospital with the diagnosis of relapsing pneumonia. The cough persisted for nine days even after the temperature was low. The patient was then radiographed and a screw was found in the right lung at a point about opposite the seventh rib. An attempt was made at bronchoscopy, but it was not successful. He left the hospital in two weeks and was comparatively well up to July, 1911, but still had slight cough and occasionally coughed up blood. This gradually became worse, the expectoration becoming foul smelling and purulent. There was slight temperature in the afternoon.

Physical examination shows dullness at the right base and near the spine of an area over which there was bronchial breathing and rales.

The X-ray examination at this time still showed the screw, point down near the eighth rib posteriorily. The child had been bronchoscoped several times and attempts had been made to remove the screw, but were unsuccessful. The foreign body had gradually moved downward in the bronchus and now lies in one of the secondary bronchi in the posterior portion of the right lower lobe, about opposite ninth rib (Fig. 16). Of late the child has been gaining weight and looks well, but coughs at times severely and vomits almost daily after coughing. No blood expectoration is present.

For the history of the above case I am indebted to my associate, Dr. I. Landsman, through whose courtesy I was permitted to examine and study the case.

Case XVI.—Isadore B., aged 2½ years, was admitted to Beth Israel Hospital in the service of Dr. F. Huber, with the following history:

Six months before admission the child had measles and pneumonia. From that time on, there has been more or less continuous cough, which has gradually grown worse, the paroxysms becoming frequent, very severe and associated with much dyspnea. Four weeks before admission the child became ill with chills, fever and rapid breathing. The severe coughing paroxysms were accompanied by purulent expectoration and frequently followed by vomiting. There was no hemoptysis. The child's condition gradually grew worse, the expectoration became fetid, there was considerable sweating, marked prostration, and great loss of flesh and strength. Upon admission the diagnosis of abscess of the right lung with localized gangrene was made and in order to exclude a foreign body as the causative factor of this condition, the patient was radiographed. The examination demonstrated the presence of a nail in the right bronchus and trachea and also the presence of a lung abscess (Fig. 17). The history was now elicited that some eight months before the child while playing with some nails suddenly began to choke, gasp for breath and had several severe paroxysms of coughing. The urgent symptoms soon subsided, but the child was left with a cough which persisted for several weeks and then disappeared. The ingestion of a nail was suspected, and the child had been examined by several physicians, but the absence of further symptoms had allayed the anxiety of the mother and this entire occurrence had completely passed out of her mind and did not appear to her nor to those under whose observation the child came for treatment of its pulmonary condition, to have any bearing upon the child's condition. The nail was removed through a tracheotomy wound. The abscess of the lung was treated in the usual way, and though tedious, the recovery of the child was complete.

Case XVII.—Jacob M., 9 years of age, nativity Russia, was admitted to Dr. F. Huber's service at Beth Israel Hospital, with the following indefinite history:

With the exception of measles as an infant, he had enjoyed fair health up to the present illness. For the past five weeks the boy had gradually lost flesh and strength. He suffered from paroxysms of coughing attended with the mucopurulent expectorations and followed by vomiting. No night sweats or hemorrhages. Occasionally he complained of chilly feelings, but no fever, with the exception of a period of three or four days, two days after the onset of his illness. The appetite was poor and pulse and temperature were normal.

Physical examination revealed retraction of the infraclavicular region on the right side with diminished expansion. The right anterior aspect of the chest was flattened. On percussion there was marked dullness over second, third and fourth interspaces anteriorily, apex not being involved. The breathing was diminished and on deep inspiration fine rales were heard over the dull area.

The X-ray examination I made revealed a dense infiltration of the upper right lobe; compensatory emphysema of the base. Enlarged glands were visible,

at the root. In the midst of the consolidated area, apparently lodged in the right primary bronchus, a tack, about three-quarters of an inch long, was detected (Fig. 18).

In the light of these findings, a careful questioning elicited the following points:

Five weeks before admission, the child accidentally swallowed a brass tack. Almost immediately he was taken with a severe coughing spell, which lasted for several minutes, the attack was not attended, however, with any evidence of asphyxia. Fearing punishment, he had failed to tell his parents about the accident.

The tack was removed by forceps directly through a tracheotomy wound, under direct vision by means of fluoroscope, a method I believe far superior, in some cases, to bronchoscopy.

In the five cases cited there was no history of ingestion, and cases were first observed when secondary symptoms, due to the long retention of the foreign substance, were present.

The problem of diagnosis in these cases is difficult, but it is usually solved

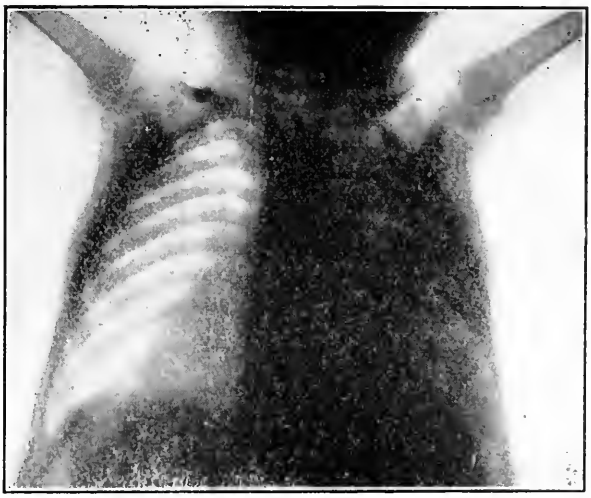

Fig. 17.—Case XVI. Nail in right bronchus and trachea, abscess of lung.

when that mental attitude is reached which suggests a radiographic or intrascopic examination. A history of paroxysmal cough extending over a long period of time gradually increasing in severity and later accompanied by chills and fever; the cough associated with profuse expectoration which becomes purulent and then putrid, progressive interstitial pneumonia with its sequelae and complications are not commonly considered to bear any causative relation to the presence of a foreign body in the bronchi. Yet by keen analysis of the subjective and objective data and the careful exclusion of such other causes as might give the clinical picture, the diagnosis of foreign body may be made even in the absence of a history entry and before the corroborative aid of X-ray examination has been given. This was done in a case coming under the care of Dr. F. Huber. In one case a child 21 months was presented in a septic bronchopneumonia and localized gangrene. The story was elicited that five months previously the child while sitting on the floor, suddenly had an attack of suffocation which soon passed away but left the child with a paroxysmal cough. Based upon this story and

the objective findings, the diagnosis of foreign body in the bronchus was made which the radiograph proved to be correct, disclosing a bent nail in the left bronchus.

A diagnosis is not, however, always made. Fatal cases are not uncommon, though less frequent in these days than they were formerly, in which foreign bodies have been found in pulmonary abscess where no suspicion of their presence existed during the life of the patient. Two interesting cases of this sort were reported by Bayer (57). In one a molar of the first dentition was found in a fatal case of abscess of the right lung and in another case the pulmonary condition was due to a sequestra from syphilitic disease of the palate.

2. There is a class of cases in which the history of entry is clear but the mildness of the early symptoms leads to the erroneous diagnosis of a foreign body in the alimentary tract, the true nature of the condition being discovered when seeking the cause of the retention of the substance within the organism.

Case XVIII. Hattie H., aged 7 years, was referred to me by Dr. Israel Strauss, with the object of locating the exact position of a pin which the child was supposed to have swallowed.

The history was that five days ago, the child, while holding the pin in her mouth and pursuing another child, suddenly stopped and cried out that she had swallowed the pin. She coughed a little at first, but in a few hours the cough ceased and the child was in every way comfortable. There was no change of voice, no dyspnea, nor any difficulty in deglutition. The treatment was based upon the assumption that the pin had been swallowed, but the carefully watched stools did not reveal its presence.

The case seemed clear. The only doubtful point being the exact location of the foreign body in the alimentary tract. Radiographic examination (Fig. 19) showed the pin in one of the secondary bronchi of the posterior part of the right lower lobe. Examination of the chest at this time showed a normal percussion note over both lungs anteriorily and posteriorily. At the right base inspiration was diminished in intensity, high pitched with a musical quality. There were no rales and tactile fremitus was unchanged. The child was admitted to Mt. Sinai Hospital and six days after the accident the removal of the pin was attempted by means of lower tracheobronchoscopy, but the efforts were unsuccessful. The child recovered from the pneumonia which followed, the tracheal wound healed, and four years after its entry the pin is still in the bronchus. The child is at the present time perfectly well with no pulmonary symptoms of any kind.

Tillman reported a case whose outcome was similar to this, the patient being a girl of five years who had aspirated a brass tack into the left bronchus. Removal by bronchoscopy was impossible and further operative measures were refused. Notwithstanding this, however, the child remained perfectly well.

Diagnosis.—The value of the history in the establishment of the diagnosis has already been discussed. Though the subjective examination of children yields but little accurate information because of their inability to definitely locate and describe sensory impressions, it is nevertheless important to weigh any evidence submitted. The story of a spasm of suffocation may give a clue to the establishment of the etiological factor of an obscure intestinal or pulmonary condition. The symptoms which are mild in the majority of cases are none of them pathognomonic, and the objective examination is therefore essential in order not only to verify or exclude the tentative diagnosis but also to obtain accurate and detailed information regarding location, position, etc., of the foreign substance, when it is definitely known to be present in the organism.

Examination of the Pharynx.—The necessity of the examination of the pharynx by direct view with the aid of a tongue spatula and in good light need not be emphasized. The tongue should not only be depressed but pulled forward as well. As illustrating the importance of this examination is the case reported by White (52) in which a pin lay in the throat of a child 18 months old for eight months. The throat had been repeatedly examined by others and nothing found. White by depressing and pulling the tongue forward found the pin lying across the pharynx just behind the tonsils.

Exploration.—Examination by direct touch of pharynx and upper air passages is not only of but little value because of the limited reach of the finger and the probability of mistaking normal structures for foreign substances but is fraught with much danger because of the possibility of displacing the substance by blind manipulation. A body resting in the glosso-epiglottic fossa, from whence its removal is a simple matter, may, during such an examination, be pushed into

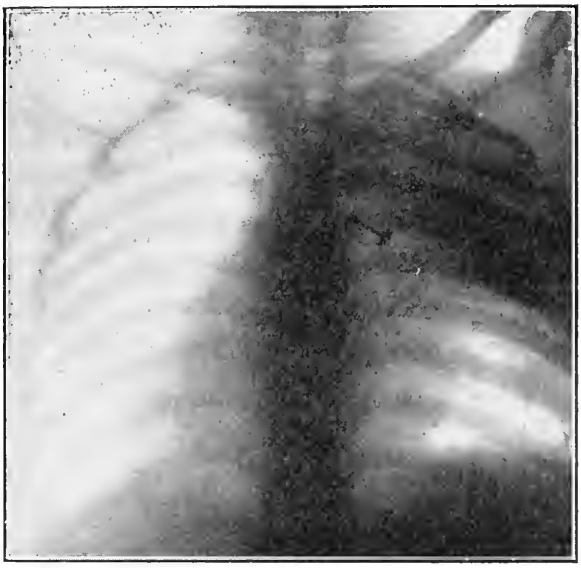

Fig. 18.—Case XVII. Tack in right bronchus.

the larynx or esophagus. The posterior pillar of the fauces has been mistaken for a fish bone. Atlee (53) quotes Nelaton, who confessed that after pushing deeply into the pharynx in search for a foreign substance he felt a small resisting body and made several attempts to seize it with forceps before discovering it to be the great horn of the hyoid bone.

A rectal examination should always be made when it is known that the body is in the intestinal tract and has not made its expected appearance in the stools. Such bodies are often found lying just above the sphincter and may frequently give no symptoms until their attempted expulsion. A boy of six years appeared in the clinic complaining of pain during defecation, with the story that the stools were usually flecked with bright blood. Rectal examination disclosed a pin with its point imbedded in the rectal wall near the anal margin. There was no history of the ingestion of the pin nor of any symptoms at any time previously. Too extensive manipulation blindly with the finger is here as elsewhere not a commendable procedure, and where the body is found fixed it is best to use a small speculum for further examination and extraction.

The examination of the esophagus by indirect touch with bougies and stilets, for the determination of the existence and consistency of the foreign body is, under the most favorable circumstances, a crude method and frequently gives erroneous results. Previous to the time when direct view of the lumen of the tube could be obtained either by the X-ray or intrascopic examination, sole reliance was placed upon these methods of exploration and considerable ingenuity was expended upon the production of instruments for this purpose. This method of examination seldom gives reliable or satisfactory results, for it is an established fact that a bougie will touch a foreign body impacted in the esophagus without sending any appreciable message to the finger or passing to one side of the tube will not betray its presence if the body does not markedly occlude the lumen. Even under anesthesia this examination does not give any more accurate

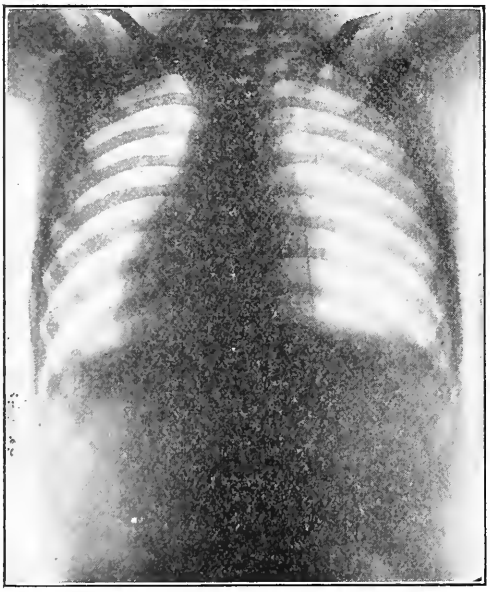

Fig. 19.—Pin in secondary bronchus, right lower lobe.

information. Should it disclose the site of obstruction, it gives no information as to other important factors regarding the impacted body. So that this method, possessing all the defects of blind instrumentation, had best be abandoned and main reliance placed upon those methods of examination which enable us to obtain a direct ivew of the foreign substance.

The examination of the esophagus by direct view by means of straight tubes inserted into its lumen is a very useful and satisfactory method. The tubes most commonly employed in the examination of children are those of Killian, Rosenheim or Jackson, and for their successful use in general, anesthesia is necessary. With these instruments the esophagus can be searched throughout its entire length and the situation, position, shape and size of the impacted body determined.

The autoscope devised by Kirstein and the pharyngoscope of Hays have been used for the supplementary examination of the pharynx and larynx. With these instruments a direct view of the pharynx, larynx, particularly the posterior wall, the interarytenoid space, and at times the trachea and the beginning of the bronchi, may be obtained. The one objection to the Kirstein instrument is that the

patient is held in a constrained position and the examiner is able to see only in the direct line of the instrument itself.

Examination of the Air Passages.—The examination of the larynx in children by means of indirect laryngoscopy is a difficult and usually impossible procedure without anesthesia. More satisfactory results are obtained by a direct view either by means of a spatula, or through a straight tube under general anesthesia with the patient in the Boyce position. Sometimes the actual introduction of the tube within the larynx is not necessary; simple pressure on the base of the tongue or on the laryngeal surface of the epiglottis with the tube spatula will disclose a view both of larynx and upper trachea.

When severe dyspnea is present laryngeal examination should not be attempted unless tracheotomy is previously done. For the examination of the trachea and bronchi the bronchoscope may be introduced, either by being passed through the larynx and trachea, superior trachea-bronchoscopy or through a tracheotomy wound inferior bronchoscopy. The previous localization of the body by the ray facilitates the examination by permitting the choice of instruments and method of introduction. Even in expert hands bronchoscopy does not appear to be an easy procedure. The small caliber of the bronchial tubes, particularly in children under four; the instability of the landmarks, particularly in a lung, in which the foreign body has been long resident; the constant movement of the entire bronchial tree, especially during the ever-present coughing; the flooding of the small bronchi with secretion, all tend to make the search with this instrument difficult, particularly if there are pathological lung changes. In cases where the foreign body has been long resident it is frequently impossible even to get a satisfactory view of the foreign body. Numerous cases have been reported in which the foreign body was expelled long after the futile bronchoscopic attempts at inspection or removal had been made.

The procedure is not without its dangers, particularly in younger children or in children with exudative diathesis. Kummel reports four cases in which direct bronchoscopy led to serious complications, with a fatal outcome in one case. Two of the patients were boys of four and a half, the others a girl of nine and a boy of twelve. In all there was a subglottic swelling which rapidly increased after bronchoscopy or developed first after it, inducing suffocation which would have proved rapidly fatal in all without the immediate tracheotomy. Even if small tubes are used this danger may be reduced but not abolished entirely. Even when practised through a tracheotomy wound this procedure is not without its dangers, as in the following case:

Case XIX. G. C., age 3 years, was examined because of a history of having swallowed a tack five days ago. The child did not look ill. There was no difficulty in swallowing. The respirations were rapid, short and grunting. The physical examination was negative except for a small area of dullness at the angle of right scapula with diminished breathing.

A radiograph showed a tack in the right bronchus and a basal pneumonia. After a low tracheotomy a bronchoscope was passed. During the manipulation before the foreign body was seen the child suddenly grew cyanotic and died.

Examination With the Roentgen Ray.—The radiographic examination plays, and must continue to play, an important part in the determination of the presence of a foreign body in the organism. It should be understood, however, that though it has considerable value as a diagnostic agent and has certain advantages in the ease and facility with which the examination is made, this method also has its limitations. There are some foreign bodies which will escape detention if this be the only examination made. In the great majority of cases, however, it allows the detection of the foreign substance and permits the study of the progress of

the condition. It permits the study of the movement of a foreign substance and aids in the determination of the measures to be pursued for its extraction.

Technic.—Whether the patients are to be fluoroscoped or radiographed it is important that they be stripped of every vestige of clothing. The examination should not be made in bed but upon a table suited for such examinations. These precautions may seem of trivial importance but they are very essential and many curious errors have arisen from failure to observe them. Rotch spoke of a child who was supposed to have swallowed a pin. In radiographing the child a little flannel band about its abdomen was not removed. The picture showed the pin and preparation for operation were made, but when the child was bathed it was found that the pin had not been swallowed but had slipped behind the band. A metallic substance lying behind the sensitive plate subjected to exposure will give a faint shadow and be mistaken for a body within the organism. A child supposed to have swallowed a safety-pin was radiographed and the picture showed a faint but distinct shadow of the pin evidently in the intestine. Later it was found that the shadow of the pin on the plate was not due to the one within the body of the child but to one which lay in the sheet upon which the photographic plate had been put during exposure.

For fluoroscopic examination the child may be either in the erect posture, being supported on either side or lying prone, the tube being placed beneath the table. With open or shaded screen the entire body from occiput to symphysis should be examined irrespective of the clinical diagnosis. The liability to erroneous diagnosis will be minimized if radiographs are made even when the fluoroscopic examination is considered satisfactory.

With the suitable apparatus and a large current it is possible to make radiographs with an exposure of a fraction of a second, and during this short interval even the most restless children may be kept quiet. A child usually very uneasy will often lie perfectly quiet the instant the tube lights up, being evidently fascinated by the glowing bulb. In many thousand radiographs made for this and other conditions, I have never found it necessary to administer an anesthetic in order to obtain satisfactory pictures. The neck, chest and abdomen should be radiographed if the body has not been previously localized with the fluoroscope.

The vast majority of the foreign substances which give troubl within the alimentary or respiratory passages of the child will cast a definable shadow upon the photographic plate if the exposure be correctly made. Occasionally a soft or thin substance in the esophagus will cast a shadow which may be lost in that of the spinal column. In those cases a radiograph made with the patient in the oblique position will throw the shadow of the esophagus away from the column and an impression of such a substance will be obtained which would otherwise not be recognizable. The dried plate placed within an illuminating box where it is possible to vary the intensity of the light, should be examined with great care and thoroughness with the eye in not too close proximity of it.

The skill required for the accurate interpretation of an X-ray plate can be obtained only from the extensive reading of such plates, normal and pathological. The actual making of the radiograph is comparatively simple and is after all only the means to an end and that is its interpretation. Every impression upon the plate is a shadow, true or distorted of some structure of or in the body and is capable of interpretation as such. A foreign body like a pin in the intestine, and thus at some distance from the photographic plate, will not only cast a distorted shadow but because of the peristaltic movement of the gut, the impression upon the plate will be hazy and indistinct as well, unless the exposure be short. The shadow of a disc-like foreign substance may appear upon the plate as a mere

line. In a case of a boy of 10 years, who had inspired a prune pit, the radiographic examination in several hospitals failed to show the shadow of this, which was, however, well defined in a plate made later.

In another case where I was able to obtain a distinct shadow of a linear foreign substance (evidently wood) in a child suffering from interstitial pneumonia, operative measures were refused because an examination made elsewhere, and made improperly, did not disclose any shadow.

No difficulty will be experienced as a rule in deciding from the radiograph whether the foreign body is in the trachea or esophagus. If such doubt exists the matter can be easily cleared up by a radiograph made in the lateral or oblique position or stereoscopically. A knowledge of the topography of the bronchi, according to their projection upon the radiographic plate, will materially assist in the extraction with the bronchoscope. Reference to stereoscopic pictures made with the bronchi of a cadaver injected with bismuth may aid in the localization of the foreign body and in the estimation of its position in the bronchial system.

Finally it must be emphasized that the examination with the Roentgen ray, using short exposures, is a harmless procedure. That the rays have a profound effect upon metabolism is not to be doubted, and yet after radiographing thousands of children suffering from every sort of a pathological condition, detrimental effects which by the most liberal interpretation could be ascribed to the examination are as yet to be demonstrated.

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MALARIA IN AN INFANT FIVE MONTHS OLD, SIMULATING VON JAKSCH ANEMIA.*

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Malaria in infants is probably not rare; undoubtedly there are many infantile cases that are not recognized. The blood findings simulating von Jaksch anemia (pseudoleucemia of infancy) and the age of the patient are the reasons for reporting this case.

History: No history of syphilis or tuberculosis in the family. Parents and three other children living and well. The patient, a male infant, five months old, was admitted to the hospital on August 1. The mother said the child was normal at birth and labor was normal; birth weight unknown. Was breast fed, but during the three or four weeks previous to admission the child was given two bottles daily containing flour and water. Child has never been out of New York City. No illness until the present, which began about three weeks ago. Mother gives the ordinary indefinite history of gastrointestinal disturbance, with slight cough. Constipation was more often noticed than diarrhea. Laxatives seemed to help at first, but during the week before admission he became weaker and the mother noticed that his color was yellowish. As he seemed to be "failing" he was brought to the hospital.

Physical examination: Male infant, fairly well nourished, but very anemic in appearance. Lies rather listlessly in bed. Head: Nothing abnormal found. Pupils normal in reaction. No jaundice. Ears negative. Mucous membranes of mouth and pharynx appear almost bloodless. Thorax: Lungs, normal resonance throughout. Heart, rapid; not enlarged; no murmurs. Abdomen: Liver not palpable. Spleen markedly enlarged; firm, not tender; extends downward to level of umbilicus and inward to within 2 inches of median line. Extremities normal. Lymph nodes: One small node palpable under right side of jaw. Skin, waxy-yellowish. Weight: 11 pounds 8 ounces, on admission. Blood: August 1, red cells, 1,036,000; white cells, 30,000; hemoglobin, 20 per cent. Differential: lymphocytes, 68 per cent.; polynuclear neutrophiles, 30 per cent.; mononuclears and transitionals, 2 per cent.; five normoblasts found while counting 500 leucocytes; no eosinophiles were found in any of the counts; no malarial organisms found.

August 5, red cells, 1,800,000; white cells, 18,600; hemoglobin, 25 per cent. Differential: lymphocytes, 72 per cent.; polynuclear neutrophiles, 24 per cent.; mononuclears and transitionals, 4 per cent.; no malarial organisms found. August 10, red cells, 1,500,000; white cells, 18,000; hemoglobin, 20 per cent. Differential: lymphocytes, 64 per cent.; polynuclears, 33 per cent.; mononuclears and transitionals, 3 per cent.; malarial organisms numerous.

On account of the severe anemia it was thought necessary to get the child under the influence of quinine as rapidly as possible; so the bimuriate was given hypodermically, four grains daily in divided doses. As may be seen by the temperature chart, there was 104° fever the day after the quinine was begun, and

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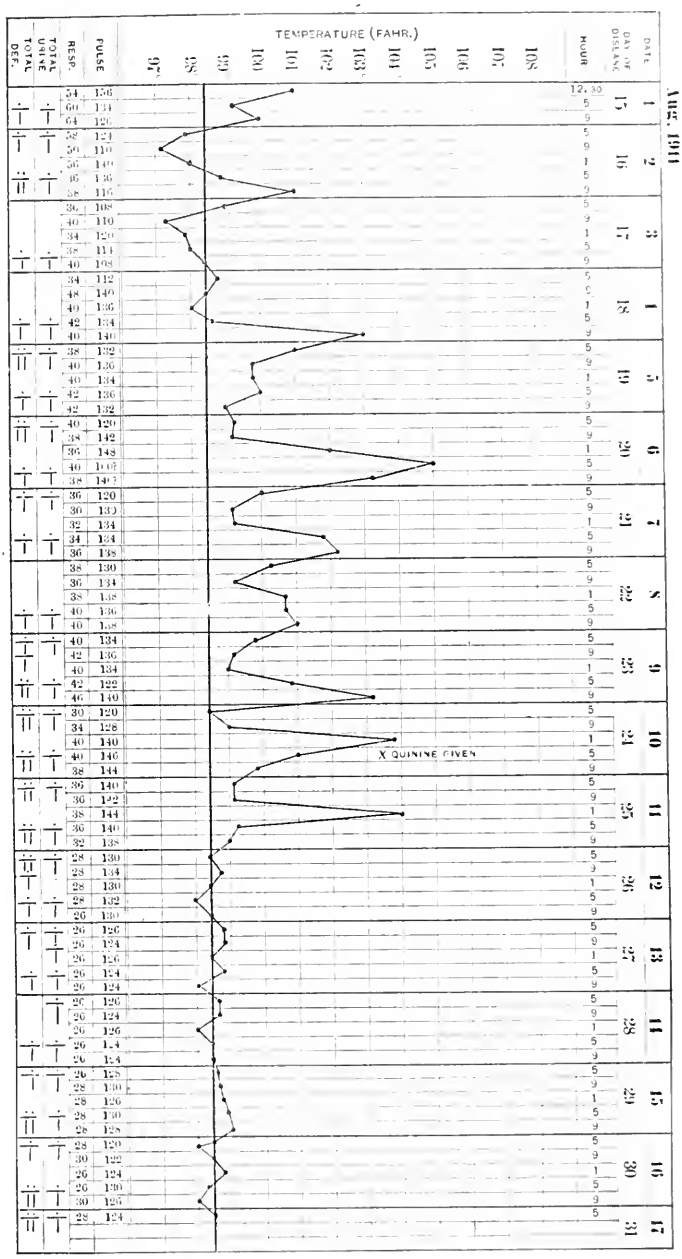

then a practically normal temperature. The child made an uneventful recovery. The decrease in size of the spleen was especially noticeable; ten days after quinine was given the spleen could hardly be palpated. The case was first diagnosed as pseudoleucemia of infancy, but the finding of malarial organisms of course indicated the correct diagnosis and treatment.

Several authorities maintain the pseudoleucemia of infancy is not a clinical entity. They hold that such cases are cases of anemia of various origins (syphil-

itic, rachitic, or after wasting diseases) and are mere examples of the manner of response of infants to severe anemias. In support of this it must be remembered that (1) the normal blood count of infants resembles the leucemic count of adults in the excess of the lymphocytes over the polynuclears; (2) the normal hemoglobin percentage of infants is lower than that of adults, and (3) the spleen in infants readily becomes enlarged during any severe anemia. The foregoing case is of interest in showing that the malarial organism may be the etiological factor in an anemia of this kind.

THE EMPLOYMENT OF SALVARSAN IN INFANTS AND YOUNG CHILDREN.*†

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If we consider only the moderate grades of congenital syphilis, the treatment of the disease by the older methods with the various preparations of mercury and iodid of potash has been quite satisfactory. When, however, we consider the severer types, particularly those showing a tendency to hemorrhage during the early days of life, and when, moreover, we contemplate the possibility of the appearance during the latter part of the first decade of the tardy manifestations of the disease, a feeling of dissatisfaction must come to us. On account of the high mortality of the serious cases, on account of the possibility of late manifestations of the disease in the eye, ear, bones or nervous system, there has long been needed, even in the management of lues in infants, some other more powerful and more certain remedy.

Moreover, since the introduction of the Wassermann reaction for the detection of the disease and also for determining that the disease has been checked, if not eradicated, there has been a still further incentive toward the use of other than the mercurial treatment. It was Ehrlich's hope that in "606," or salvarsan, he had obtained the great sterilizing agent which would by one administration, if in sufficient dosage, entirely destroy the spirochetae and so rid the system of the disease and of its consequences.

The use of salvarsan in adults has been so general during the past two years that very definite conclusions have been reached in regard to technique and dosage. With regard to the use of salvarsan in infants, however, there is not as yet the same certainty in this respect.

The salvarsan may be administered to the infant indirectly by injection of the pregnant or of the nursing mother, or it may be given directly to the infant.

According to the conclusion of De Buys (1) the results on the infant of injecting the pregnant woman with salvarsan have been generally unfavorable. He quotes Gluck as having injected a woman seven months pregnant with resulting death of the fetus on the following day.

Good results from injecting the nursing mother have been reported by Taege (2), Duhot (3), Malinowski (4), Dobrovits (5), Sequeira (6), Marschalko (7), and others. Negative results from the indirect method are reported by Peiser (8), Rosenthal (9), Ritter (10). Oppenheim (11) and Spiethoff (12) report

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negative results by the injection of the mother, with later good results after direct injection of the infants.

The best and most rapid results are obtained by the direct injection of salvarsan into the infant. The routes for injection have been the subcutaneous, the intramuscular and the intravenous. The subcutaneous injections produced bad sloughs or cellulitis and so were soon abandoned. The intramuscular (usually intragluteal) injection of a parafin oil emulsion of salvarsan in dosage of 0.015 gram per kilogram is favored by Hochsinger (13) and others, while von Bokay (14) prefers Wechselmann's neutral suspension in dosage of 0.008 to 0.01 per kilogram. Von Bokay used this method in 26 cases, giving second injections in ten instances, and seems enthusiastic over the direct method, though the occurrence of three relapses leads him to the conclusion that the ideal procedure with salvarsan has not yet been determined.

Savariaud (15) reports a good result by direct treatment of a girl of four years suffering from bone lesions after mercurial treatment had been unavailing.

Among others who reported good results from the direct method are Escherich (16), 6 cases; Lesser (17), 9 cases, and Miekley (18), 5 cases.

Toxic polyneuritis from the intragluteal injection of 0.3 gram in an eighteen months old girl has been reported by Fischer (19), and parenchymatous nephritis along with necrosis of the subcutaneous fat, muscles, blood vessels and nerves has been reported by Merkel (20) as the result of the intragluteal injection of salvarsan in a two-and-one-half months infant.

During 1910 and 1911, there have been treated on my service in the children's wards at Bellevue Hospital 25 cases of hereditary syphilis of the congenital type. Of these, 10 received salvarsan either with or without mercurial treatment, while 15 were treated by the use of mercurials alone. It should be said in explanation of the large number that did not receive salvarsan treatment, that some of these patients were in a moribund condition and died within two or three days after entering the hospital, even before a Wassermann reaction could be obtained; others were such mild cases that it seemed wise to use only mercury; while for still others there was objection on the part of the parents to the administration of the new remedy intravenously.

The ages of the 15 patients in whom only mercurial treatment was used ranged from three weeks to one year—the most of the patients being about three months old. The ages of the 10 salvarsan cases ranged from two months to five and one-half years.

The two sets of cases were treated in the wards side by side without selection, save that the moribund cases were not given salvarsan.

The symptoms of the infants and younger children were quite similar in the two groups of cases, with the exception that the one patient five and one-half years old receiving salvarsan had severe bone lesions.

The mercurial treatment consisted usually in the administration of gray powder ¼ to ½ grain two or three times a day, together with inunctions of 25 per cent. blue ointment every second day. In some cases the salicylate of mercury was injected intramuscularly in doses of 1-10 of a grain every second day.

The technique of the salvarsan administration was as follows:

Only freshly distilled water, heated up to 120°F., was used in making up the solution.

The contents of the ampulla of salvarsan, containing 0.6 gram of the drug, are poured into 100 c.c. of the hot water and then shaken thoroughly until dissolved. Four per cent. NaOH solution is then added cautiously until the fluid is clear. It usually takes about 0.7 c.c. of the 4 per cent. NaOH solution to

each 0.1 gram of salvarsan, or about 4 to 4.5 c.c. of the NaOH solution to alkalinize the 0.6 gram salvarsan in the ordinary ampulla. Then more hot distilled water up to 300 c.c. Each 50 c.c. of this solution represents 0.1 gram, or each 5 c.c. represents 0.01 gram of salvarsan, which is convenient for dosage. The cloudiness often found in the salvarsan solution is filtered out through sterile gauze. Now the solution is ready for injection. It is most convenient to make use of an apparatus devised by Dr. Albert M. Meads, of Bellevue Hospital, New York, who has, since December, 1911, given 43 injections with it. This consists of two tall 300 c.c. vessels, one for the salvarsan solution and the other for salt solution, connected by tubes to a Y, from which passes the long tube carrying the needle for injection. Both vessels are connected at the top with a tube leading to a pressure bulb which can be used to hurry the liquids out of the vessels. When the patient's vein is ready, some 0.9 per cent. NaCl solution is run through the long tube. Next, plunge in the needle and run in a little NaCl solution, then turn stopcock and run in the desired amount of salvarsan solution. Then run in more NaCl solution to force the salvarsan further into the general circulation, and out of the vein.

There is seldom any rise of temperature afterward, in most cases none at all. At times a rise of temperature the same day may be due to the mere excitement of the operation itself.

Because of the disastrous or unsatisfactory experiences of others, none of the salvarsan was given intramuscularly or subcutaneouly. It was always given intravenously and nearly always after having cut down and exposed a vein at the bend of the elbow. To plunge the needle through the skin and subcutaneous fat and into the vein is not easy in infants, since the vein slips away or else is pierced clear through. Recently it has been proposed to use the vein of the scalp, and no doubt this will simplify the administration in many cases.

The ages, the weight of the patients and the dosage employed were as follows:

Case	1,	2 mos	Weight, 9 lbs., or about 4½ kg.	Dose, 0.0. gram
Case	2,	9 mos.	Weight, 7-8 lbs., or about 3¾ kg.	Dose, 0.05 gram
Case	3,	3 yrs.	Weight, 25 lbs., or about 12 kg.	Dose, 0.10 gram
Case	4,	2 yrs.	Weight, 23 lbs., or about 11½ kg.	Dose, 0.10 gram
Case	5,	2 yrs.	Weight, 23 lbs., or about 11½ kg.	Dose, 0.08 gram
Case	6,	3 mos.	Weight, 9 lbs., or about 4½ kg.	Dose, 0.05 gram
			on Octiber 12th, and second dose, December 18th,	0.05 gram
Case	7,	5½ mos.	Weight, 27 lbs., or about 13 kg.	Dose, 0.10 gram
			on October 22d; second dose, January 7th, 1912,	0.10 gram
Case	8,	17 mos.	Weight, 8 lbs., or about 4 kg.	Dose, 0.10 gram
Case	9,	2 mos.	Weight, 17 lbs., or about 8 kg.	Dose, 0.10 gram
Case	10,	11 mos.	Weight, 9 lbs., or about 4½ kg.	Dose, 0.10 gram

These last two doses were nearly double the proportions used before.

Reaction to the Intravenous Injection.—As regards local or general reaction from the intravenous injection, there was never any necrosis or inflammation at the site of venipuncture, and the febrile reaction spoken of by many authors was seen only three times, even then being very mild, a rise on same day to 103°F. in 1 case and up to 101° or 102°F. in 2 cases on the second or third day after injection. The absence of febrile reaction is thought to be due to the use of freshly distilled water for making up the solution.

In 1 case in which there had been an irregular fever up to 102½°F. before the injection, the temperature gradually came down to normal after injection, coincidently with the disappearance of the snuffles and the fading of the maculopapular eruption.

The results of the 15 cases treated by mercurials alone were as follows: Three improved, 2 unimproved and 10 died. The 10 cases treated by salvarsan showed a mortality of only 2; all the other cases were decidedly improved and

several showed marked gain in weight and improvement in general condition in addition to the disappearance of their specific symptoms.

One child two years old gained 5 pounds in weight in two and one-half months, though the Wassermann remained positive until time of discharge from the hospital.

The five and one-half-year-old child gained in weight from 27 to 36 pounds during her six months' stay in the hospital, having had two salvarsan injections and later protoiodid of mercury pushed to the limit of tolerance. Another child,

Fig. 1.—Photograph showing saddle nose and marked swelling of radius and ulna of left forearm.

seventeen months old, gained nearly a pound in her short sojourn of nine days in the hospital.

The detailed record of the five and one-half-year-old child seems worth reporting, since it showed a relapse of symptoms while under observation and most marked improvement by the employment of both salvarsan and mercurial treatment.

Jeannette K. Five and one-half years old. Admitted October 12, 1911. Discharged improved April 9, 1912.

Family history of 2 still-births and of 2 other children dead of convulsions.

Previous personal history not obtainable further than that the child had suffered from painful swollen wrists and joints for some months. Both wrists were swollen, together with the bones just above the wrist, and on the right side there was a tender fluctuating swelling.

Saddle nose, enlarged spleen, liver and lymph nodes, together with some swelling of the bones of legs completed the picture of late hereditary lues.

The photograph taken November 1st shows fairly well the surface appearance of the forearms. (Fig. 1.)

Fig. 2.—Radiogram of left forearm, showing the thickening and destruction of lower ends of radius and ulna.

X-ray pictures taken two days after admission showed characteristic bone changes—subperiosteal infiltration with osteoporosis especially marked at the lower ends of the greatly thickened ulna and radius. (Figs. 2 and 3.)

On October 15th, three days after admission, the Wassermann reaction was found to be positive, though only weakly so.

On October 22d, 0.10 gram salvarsan was given intravenously, and that same night there was a temperature reaction up to 103°F. This lasted only a few hours, and on the following day the temperature was normal. The tenderness and swelling in the forearms improved rapidly, but on December 5th the X-ray

Fig. 3.—Radiogram of right forearm, showing thickening of shaft of radius.

showed that there had been only slight improvement in the condition of the bones.

On December 11th the Wassermann test was still positive.

On January 5, 1912, on account of a very definite relapse of the forearms to a condition almost as bad as upon admission to the hospital, a second injection

of salvarsan was given—the same amount as at first, 0.10 gram, was injected intravenously.

This time there was absolutely no temperature reaction. Now in addition to the salvarsan injection protoiodid of mercury, 1-10 grain t. i. d., later increased to

Fig 4.—Radiogram of right forearm, taken four months after Fig. 3, and showing improvement in shaft of radius.

1-5 grain t. i. d. This was continued until mild symptoms of salivation showed themselves, about March 23.

The improvement after the second injection, and coincident with the administration of protoiodid was rapid and continuous, so that the bones of forearms

and legs became normal to ordinary physical examination. The Wassermann reaction became negative and was still negative on May 20th. Unfortunately, the child was removed from the hospital unexpectedly, so that I am unable to exhibit X-ray pictures showing the condition upon discharge. The improvement in the condition of the right forearm is shown in Figure 4.

The case would seem to indicate, however, (1) that the initial dose was too small, and (2) that supplementary treatment by mercury and iodids is of great advantage.

The Wassermann reaction before the salvarsan injection cases was negative in 2 cases, negative in another on October 4th, and positive on October 20th; positive in 3 besides the alternating case just mentioned and not obtained or doubtful in 4. The clinical features of the disease were perfectly typical in the cases that had a negative Wassermann.

After the injection of salvarsan in the dosage mentioned, the Wassermann was found to be still positive in 2 out of the 3 cases in which the reaction was tested. In both these cases the clinical symptoms of the disease had entirely disappeared.

In only 1 case, that of the child five and one-half years old, did the Wassermann reaction become negative, and from the reports of other observers there is no assurance that this disappearance is permanent.

Before closing I wish to express my thanks to my resident physician, Dr. Miner C. Hill, who carried out ably and skilfully the details of treatment; to Dr. I. Hirsch for the excellent radiograms, and to Dr. Gus R. Manning, who assisted me in looking up the literature.

Conclusions.—(1) While the indirect method of giving salvarsan to the nursing mother is valuable and should be used when the mother is available, the surest method consists in giving the salvarsan to the infant. Both indirect and direct administration should be employed whenever possible.

(2) The intravenous route of administration is the best. Usually it will be found easiest to expose the vein before attempting to insert the needle.

(3) The dosage should be not less than 0.01 gram per kilogram of body weight.

(4) Repeated injections and supplemental treatment by mercurials may be necessary.

(5) The Wassermann reaction should be followed for a year.

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AN EARLY CASE OF CHONDRODYSTROPHY WITH RADIOGRAM AND NECROPSY.*

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History.—The subject of this report, R. B., was admitted to the Infants' Ward of Bellevue Hospital, Nov. 1, 1911, five hours after birth. She was said to have been born at full term, and was delivered without difficulty by a midwife. As to the family history, there had been three other children, all of whom were dead, one at ten months and the other two, which were twins, at 2 months. No miscarriages nor stillbirths. There is no history of any similar deformities in the family and there are no dwarfs in the family connection; also no history of tuberculosis, syphilis nor any other constitutional disease.

Physical Examination.—Well nourished infant, the general appearance of the trunk resembling a seal. The skin is very cyanotic and the most striking feature about the face is the marked exophthalmos. The nose is short and saddle-shaped, with a deep depression at its root. The soft palate is fissured up to the posterior margin of the hard plate. The tongue is very large, filling up the entire posterior pharynx.

The heart sounds are normal; no murmur.

The liver is palpable at edge of costal margin; the spleen is not felt.

All the bones of the extremities are exceedingly short and their diameter is comparatively large; the flat surface of the long bones is wider than normal and the bones themselves are bowed, the normal curves being greatly exaggerated. The bowing is most marked at the epiphyseal ends of the bones.

The humerus is very short and the finger tips reach only to the crest of the ilium.

The left tibia is so short and so bent on itself that one's forefinger, if laid on the internal surface of the bone, is found to be simultaneously in contact with the upper tuberosity, with the shaft and with the internal malleolus.

Measurements: Head, circumference, 41.5 cm.; bitemporal diameter, 9 cm.; biparietal diameter, 11.3 cm., occipito-frontal, 12.7 cm.; occipito-bregmatic, 10.7 cm.; occipito-mental, 13.5 cm.; anterior fontanel, 6.3 cm.; posterior fontanel, 1.5 cm. long; bisacromial diameter, 12.7 cm.

Length: Crown of head to umbilicus, 25.2 cm.; umbilicus to sole of foot, 15.0 cm.; acromion-olecranon (arm-length), 6.3 cm.; diameter, 9.3 cm.; olecranon-wrist (forearm-length) 4.5 cm.; diameter, 8.3 cm.; hand, 5 cm.; diameter, 7.5 cm.

Lower Extremity: Anterior superior spine to condyle, 7.5 cm.; circumference, 13.0 cm.; condyle to ankle, 7.5 cm.; circumference, 9.5 cm.; foot, 6.3 cm. long; diameter, 7.5 cm.

The thorax at nipple is 26.5 cm. in circumference; expansion is about 2 cm. December 19, the hemoglobin was 65 per cent.; red cells, 5,700,000; white cells, 17,400.

While in the hospital the baby was fed almost entirely on breast milk; but it gradually lost weight and died without symptoms of any definite disease at the age of 10 weeks. The weight on admission was 5 pounds 4½ ounces; it had fallen to 4 pounds 12 ounces before death.

*Specimen presented at the Annual Meeting of the American Pediatric Society, Hot Springs, Va., May 31, 1912.

*Reprinted from the American Journal of Diseases of Children, Vol. 5, pp. 18-24.

Radiographic examination of the whole body was made Nov. 9, 1911, by Dr. I. S. Hirsch, radiographer of Bellevue Hospital, whom I wish to thank for his skilfull work. His report is as follows:

Radiographer's Report.

"The radiograph shows all the essential bony and joint changes characteristic of achondroplasia. There are no points of ossification for the epiphyses of any of the bones of the upper extremity; but the lower epiphyses of the femora

Fig. 1.—Photograph taken at 1 week. It shows the exophthalmos, the pug nose and the very short arm and forearm. The finger tips reach only to the crest of the ilium, and the fingers are not separated to form the "main en trident."

are partially ossified. The diaphyseal portions of the bones are broadened and sharply outlined."

The X-ray print shows particularly well the shortening and thickening of the long bones. For comparison there is presented also the X-ray print of a normal though premature infant of practically the same length.

Pathologist's Report.

Necropsy.—I wish to thank Dr. Charles Norris, the Pathologist of Bellevue Hospital, for his careful autopsy, for his interest and help in the study of the case, and for permission to make an abstract of the post-mortem record as follows:

Anatomic Diagnosis.—Chondrodystrophia congenitalis; rachitis (1).

The body is that of a female child, 42 cm. long and weighing 2,270 gm. No description of the skeletal changes will be given because of the excellent X-ray photograph of the cadaver.

Fig. 2.—Photograph taken after death at 10 weeks. It shows also the marked thickening and bowing of the tibiae.

There is a very marked exophthalmos. Pupils normal. There are no external congenital anomalies. The vulvae are large and swollen. The anterior fontanelle is large, 5 cm. in width by 6 cm. anteroposteriorly. The posterior fontanelle is very small, but open. The sutures are wide. The face is of normal size. The right ear is thickened and red, showing a few superficial ulcerations on the antitragus. The left ear is normal.

There is a marked rosary, the swelling of the costochondral junctions being most marked internally. The ribs are long. There is a longitudinal groove just

Fig. 3.—X-ray print of patient when 8 days old. It shows the characteristic shortening and thickening of the long bones.

external to the costochondral junctions, but there is no Harrison's groove. The small intestines are markedly distended with gas. The peritoneal cavity contains blood-stained fluid (possibly due to post-mortem changes and freezing, the body having been frozen). The urachus is persistent for a distance of about 1 inch. The cord is normal. There is a small umbilical vein. The sigmoid flexure is very long, extending well into the right side. The mucosa of the small

and large intestines is everywhere very smooth, there being no solitary follicles or Peyer's patches visible.

Spleen: Weight 10 gm. It is 56 mm. by 25 mm. It is firm, dark red on section. No spleen follicles are visible.

Liver: Weight 90 gm., normal in size, dark red in color; serosa is everywhere normal and glistening; section dark red and smooth, without gross lesions.

Gall Bladder: This viscus is normal.

The mesenteric lymph nodes are everywhere small and somewhat yellowish in color.

Lungs.—The lungs show normal lobulations. The left lower lobe is completely atelectatic, red, firm, smooth on section, with extreme congestion. The posterior borders of all the lobes of the right lung are atelectatic. Otherwise the lungs and bronchi are normal.

Heart: There is no fluid in the pericardial cavity. The heart is possibly somewhat enlarged. The apex is formed by the left ventricle. There is a moderate Rechtslage, the aorta being visible and to the right of the pulmonary arteries. The right heart is somewhat large. There is a small foramen ovale. The ductus botalli is large. There is no stenosis of the isthmus. All the valves of the heart are normal. Musculature of the heart normal. The aorta and the vessels given off from the aorta are normal in distribution and appearance.

Pancreas: Weighs 5 gm.; is small; the lobulations are possibly not well developed. The head of the pancreas is somewhat red (post-mortem changes?).

Adrenals: The left adrenal weighs somewhat less than 2 gm., the right 1.25 gm. The left measures 32x25 mm., the right 15x13 mm. The cortex is very thin and pale. The medulla of both adrenals is hyperemic. There is almost no perirenal fat.

Kidney. The left kidney shows slight fetal lobulation. The pyramids are normal. Weight 13 grams. The right kidney was not measured but appears to be of the same size and is similar in gross appearance.

The genital organs show no anomalies.

The thyroid gland is very small. The lobes are symmetrical. Both lobes weigh 1 gram. Thymus is small; weight 5½ grams. There appears to be almost no glandular tissue present. The fat of the anterior mediastinum is edematous. The tongue is about normal in size. Both tonsils are extremely small. The pharynx and larynx are normal. There is no hyperplasia of the lymphatic apparatus. The parathyroids were dissected out, seven being found.

Although there was no edema of the skin of the legs or arms, the subcutaneous tissues of the thorax and other portions of the body are everywhere slightly moist or edematous.

Head: Superficial examination of the brain, which was not opened until hardened, reveals no gross lesions. The dura appears everywhere to be slightly thickened. The foramen magnum is very narrow, showing marked irregularity; and the clivus is steep.

The foramen magnum is very peculiar, being distorted and irregular. The basilar portion of the right is at a level 6 mm. below the occipital, and the shelving occipital portion is obliquely placed, so that the opening for the cord is oblique, its sagittal diameter running from the left anteriorly to the right posteriorly. This diameter measures 6 mm., while the longer obliquely transverse diameter measures 11 mm. The steep clivus is 28 mm. long.

On microscopic examination the thyroid gland shows an advanced grade of interstitial thyroiditis.

There is also an infiltration of small round-cells in the subcutaneous tissue and in the muscles, those from the abdominal wall alone being examined. Similar changes have been described in myxedema.

As to the gross anatomic diagnosis of rachitis, it should be recorded that the sections taken from the costochondral junctions and from the femur show no evidence of rachitis, the lesion being characteristic of chondrodystrophy with an extreme grade of mucous degeneration of the cartilage cells.

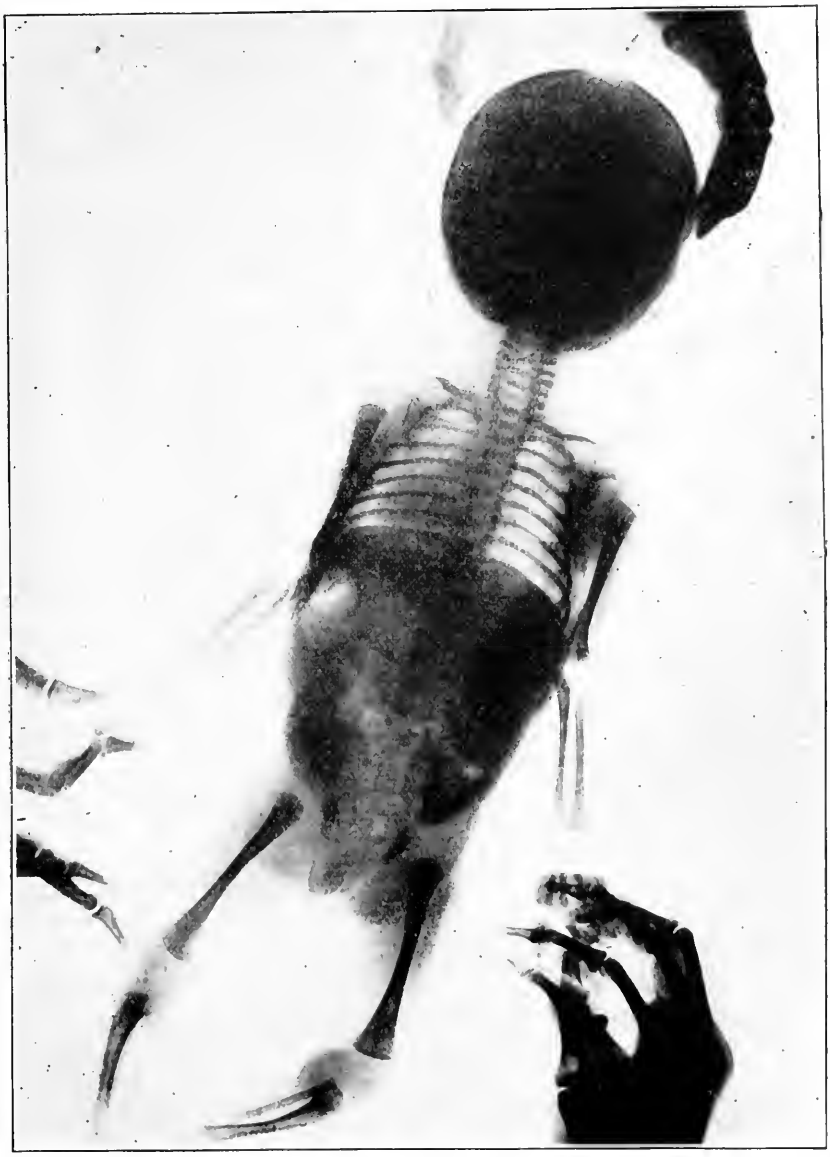

Fig 4.—X-ray print of healthy premature infant 14 days old, for comparison of long bones. The infant was 44cm. long, or only 2 cm. longer than the chrondodystrophic infant.

The case reported, while exhibiting the characteristic features of chondrodystrophy, must be regarded, in view of the gross and microscopic examination, as one of mixed type. There is no redundancy of skin on the lower extremities, there is no trident hand; there is a congenital anomaly in the cleft palate, and

there is evidence of thyroid deficiency which has probably no etiological significance. The general subject of chondrodystrophy has been so thoroughly discussed in recent literature that here no description of the disease will be attempted. For a full consideration of the subject the reader is referred to Kaufmann's monograph on "Chondystrophia Foetalis," and to the admirable volume on "Dwarfism" in the Eugenics Laboratory Memoirs XV, Section XV, a. Here will be found a complete discussion of achondroplasia by Dr. H. Rischbieth, together with a résumé by Amy Barrington of the Bibliography of Dwarfism, and reproductions or descriptions of the dwarfs (mostly chondrodystrophic) depicted by famous painters.

REFERENCES.

1. For correction of this gross anatomical diagnosis, see note in microscopical examination.

THE DIAGNOSIS OF INFANTILE TETANY

*With a Report on Experimental Tetany in Dogs.**

HERBERT B. WILCOX, M.D.

Tetany, in infants, has been recognized for many years as a disease entity evidenced by laryngospasm, respiratory spasm, muscular rigidity, contractures of the extremities, convulsions and even coma, and more recently by increased response to electrical and mechanical nervous stimulation. Many causes have been given for this symptom-complex, the earliest referring to tetany as the most dangerous of the complications associated with dentition. The first accurate description of the disease is that of J. Clark in 1815, which gives in detail the characteristic respiratory and muscular conditions. A few years later Marshall Hall wrote of the symptoms as brought about by stimulation of the central nervous system through peripheral irritation. In 1829 Kopp attributed the laryngospasm to thymus hypertrophy and at about the same time Leigh claimed that it was due to pressure on the vagus by enlarged tracheobronchial lymph-glands.

The first investigation of the electrical hyperirritability of children suffering from tetany was carried on by Escherich and von Wagener in 1890. In this and later communications (1) was established the fact that there is an increase of electrical irritability in all cas s of the disease, and that this makes its appearance before the existence of muscular spasm and is demonstrable after the latter has disappeared.

A few years after this Thiemich (2) ascertained the normal electrical irritability in young children and stated that, of the variations from this evidenced by those having tetany, the most significant one was the appearance of anodal opening contraction at a point lower than anodal closure and that of kathodal opening contraction below 5 milliamperes of current strength.

The determination of the normal response to galvanic stimulation in children and the knowledge that this became greatly increased in tetany provided a more certain method of diagnosis than had previously been at hand. Its application to large numbers of children brought out the fact that tetany was much more frequent than had been before believed. Indeed, Finkelstein (3) found that between 40 and 45 per cent. of artificially nourished children, on being subjected to the galvanic test, showed increased electrical irritability. His observations and those of Gregor, tending to show that electrical irritability was more marked in children fed on cow's-milk than in those on breast-milk or farinaceous foods, indicated that anomalous conditions of assimilation might

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produce electrical and, at times, other symptoms of tetany. This was attributed both to the greater calcium content of the cow's-milk and to the presence in this serum of an unknown substance which is lacking alike in breast-milk and in farinaceous foods.

Although it has been demonstrated that electrical response does vary with changes in digestive and metabolic processes, the fact remains that by this method of electrical tests, cases hitherto not suspected of tetany may be brought to correct diagnosis. Just what electrical findings are essential to a diagnosis of tetany has been and is still a matter of dispute. Thiemich's contention was that a kathodal opening contraction must be obtained at a point less than 5 milliamperes if tetany is to be proved. Ganghofner (4) confirmed Thiemich's findings, but stated that in many unquestionable cases of tetany the kathodal opening never dropped below 5 milliamperes, and that, moreover, other children had distinct electrical hyperirritability who never before or after had shown any signs of having tetany. Von Pirquet (5) decided from a large experience that the normal reactions of children were lower than Mann found them to be and that the appearance of anodal opening below 5 milliamperes was certain evidence of increased electrical irritability. Escherich believed that in normal children only kathodal closing contraction appears under 5 milliamperes and that only occasionally may anodal closure be present with this current strength.

My own study of the muscular response to galvanism is in two parts. The first, consisting of the routine testing of 118 infants in the children's ward of Bellevue Hospital, was instituted with the intention of adding our experience to the foregoing studies of the electrical response in children, normal in so far as their nervous systems were concerned; to determine what percentage of such routine tests resulted in distinct evidence of hyperirritability, and, if possible, to follow those children showing such reactions to see if they ultimately did develop frank tetany.

It is very difficult to define exactly the normal electrical response of childhood. Some children react low one day and high the next. Some give low closing contractions for kathode and anode, others lower anodal opening than anodal closure. It is common to obtain kathodal closure response below 2 milliamperes, while the other reactions are well above 5 milliamperes. In view of the number of our children, normal as to their nervous systems, who in this series gave reactions other than kathodal closure below 5 milliamperes, and in view of our experience with the reactions of normal dogs, it seems that Escherich's definition of hyperirritability imposes rather too narrow limits on the normal reaction, but it is, nevertheless, followed as a basis of division in the following tables.

It is well understood that definite conclusions cannot always be drawn from such classifications and averages as are hereafter given. They are, however, valuable as teaching something of the results to be expected in electrical tests in infants apparently normal in so far as their nervous systems are concerned, and do not apply to those suspected of or actually suffering from tetany.

The second part of the article, dealing with the electrical reactions in dogs, is intended to demonstrate that in normal dogs these reactions are similar to those in normal children, and to offer some observations on the relation between lesions in the parathyroid glands and electrical hyperirritability as the first evidence of the ensuing tetany.

We have to consider, then, a disease rather common in infancy and also in adult life, but in the former case presenting an entirely different symptom-complex from that of the adult type.

Potpeschnigg found the incidence of tetany to be 109 unquestionable cases

in 10,000 children observed in the Grazer children's clinic in 1900-1904, or about 1 per cent. The greatest number of cases occurred between the third and twentieth months, during this period going as high as from 4 to 6 per cent. of the children admitted between these ages. There is a wide divergence of opinion on the question of the frequency of the disease, due, presumably, to the varying attitudes of the observers as to what constitutes a true diagnosis. Various authors give figures varying from 6 per cent. down to 0.7 per cent. in artificially fed children under 3 years of age.

In Dr. Howland's service at Bellevue, of 934 children under 2 years admitted from October, 1909, to March, 1911, there were five unmistakable cases of tetany, or 0.5 per cent. Two of these were admitted before the routine electrical tests were begun and were not so tested. The three others showed marked hyperirritability to galvanism in addition to laryngo- and respiratory spasm and muscular rigidity.

The type of tetany generally presented by children of this age is that accompanied by great general irritability, laryngospasm and respiratory spasm, with or without convulsions, rather than by spasms of the extremities. The latter condition is, however, demonstrable in most cases if carefully sought for. The electrical diagnosis of tetany, the technic and details of which are later referred to, depends on the obtaining of muscular response to galvanic stimulation with kathodal and anodal closure and opening at a current strength of less than 5 milliamperes. Such electrical hyperirritability, that is, an irritability toward all four forms of current, is not present in children with normal nervous systems. It appears early in the course of tetany and, while varying greatly from time to time, persists after all other tetanoid symptoms are absent. An instance of the variation sometimes found is that of a 15 months' child who, on the first test, reacted to kathodal closure, 0.6 milliamperes; anodal closure, 1.6; anodal opening, 1.6; kathodal opening, 2.3. Six days later, kathodal closure, 2.0; anodal closure, 4.0; anodal opening, 3.0; kathodal opening greater than 5.0, and after three days gave in a third test: kathodal closure, 1.6; anodal closure, 3.0; anodal opening, 1.3; kathodal opening, 3.0.

An incomplete reaction may be frequent in cases of true tetany. This is shown in Dogs 3 and 6, neither of which gave response to kathodal opening below 5 milliamperes, although both unmistakably died of the disease. It is evident from this and other similar experience that a true decision for or against tetany can be reached only after repeated galvanic tests.

Escherich's investigation of children under 6 months of age showed 55 per cent. evidencing hyperirritability; i. e., giving some reactions other than kathodal closure with currents of less than 5 milliamperes.

In my series of 318 electrical tests, ten, or 3.1 per cent., gave evidence of extreme or kathodal hyperirritability. Of the middle grade or anodal irritability giving response to kathodal closure and anodal closure alone below 5 milliamperes, there were 101, or 31.8 per cent. With kathodal closure and anodal opening alone present, below 5 milliamperes, I found fifty, or 16.6 per cent. With both kathodal closure, anodal closure and anodal opening, less than 5 milliamperes, there were forty, or 12 per cent. The occurrence, then, in my series of abnormal reaction or increased electrical response according to Escherich's definition was 193, or 61 per cent.

Of these cases of anodal or partial hyperirritability, none developed other symptoms of tetany under observation. What then does this condition of partial irritability mean? Is it a state of exaggerated nervous sensitiveness quite unrelated to tetany and to be expected in about one-half of the children who come under observation? Is it an evidence of the past existence of the disease, or may it be the precursor of the development of tetany in its true form? The first

hypothesis would seem to be the most probable one, and if so, necessitates the definition of normal electrical reaction as a condition of irritability responding by muscular contraction to kathodal closure alone with currents of less than 5 milliamperes strength, or in an equal number of instances, to anodal closure and anodal opening stimulation with the same weak current, the former being twice as frequently found as the latter. Reason for this belief is found in the fact that the condition is common to so many children who evidently have not tetany, and strength is added to it by the demonstration of the same type of irritability on my dogs before operation.

On the other hand, and perhaps in support of the theory that this anodal irritability is the precursor of true tetany, it will be noted that in all the partially parathyroidectomized animals there was evident an increasing grade of anodal irritability. Further experiments bearing on this point are at present in progress.

Evidence of mechanical hyperirritability in tetany may be demonstrated in the following ways:

The Chvostek Sign.—This depends on reflex contractions produced in the muscles at the angle of the mouth, the ala of the nostril and brow by tapping the cheek over the facial plexus midway between the angle of the mouth and the zygomatic arch. This symptom is given varying grades of importance by different authors. It was present in but one instance in my cases, this being one of evident tetany.

Trousseau's Sign.—By shutting off, through pressure above the elbow or in the groin, the blood-supply to arm or leg, there ensues during the pressure or immediately on its release the typical carpal or pedal spasm, as the case may be. This also was present in but one case of my series.

Routine electrical tests were done during the months October to February, inclusive, and during June and July. It is unfortunate that they could not be carried on through the entire year in order that some conclusion might be reached as to the influence of season on the incidence of tetany and the occurrence of hyperirritability. In one series of 246 cases of tetany collected in one year 74 per cent. occurred during the months of January, February, March and April. The highest percentage (24 per cent.) occurred in March. Three of my cases of frank tetany occurred in February, two each in December and January. The incidence of hyperirritability was greatest in December.

It is not possible from the data herein contained to draw any conclusions as to the relation between the incidence of tetany according to season and that of hyperirritability of the lower grades. Information on this point would aid in interpreting the meaning of the anodal grade of irritability.

If we accept the classification of Escherich and consider as evidence of at least some degree of hyperirritability, the appearance of kathodal and anodal closure and anodal opening contractions with less than 5 milliamperes of current, my own figures would show that in January 23 per cent. of reactions showed hyperirritability; February, 24.5 per cent.; June, 9.6 per cent.; July, 17.7 per cent.; November, 16.6 per cent.; and in December, 27.6 per cent.

In October the number of tests made was too small to be of consequence. The number of tests made each month was: October, three; November, twelve December, twenty-nine; January, seventy-four; February, fifty-seven; June fifty-two; July, ninety.

The months of December, January and February gave the greatest number of middle-grade reactions, which corresponds in part to the months in which Escherich found the greatest incidence of tetany. Seven of my ten cases giving complete tetany reactions were under observation in these three months. Al-

though followed up to the time of their deaths, none of these ever showed other evidences of tetany. They were all cases of malnutrition and the patients are reported to have died outside the hospital from nutritional causes. These children may represent instances of increased reaction to galvanism accompanying metabolic disturbance of severe grade and not due to the tetanoid condition. They were, however, discharged from the hospital months before death and the reports made on their condition during this time are not complete enough to positively exclude the possibility of the existence of true tetany.

Three hundred and eighteen tests were made on 118 children, being repeated more frequently on those showing a tendency to electrical hyperirritability.

The instrument used supplied galvanic current from dry cells, and contained a switch for reversing its polarity, a rheostat for controlling the current strength, and a balanced milliamperemeter measuring from 0.2 to 10 milliamperes.

Ten of our cases gave low reactions with all four tests, kathodal closure, anodal closure, anodal opening, and kathodal opening. These children were suffering from varying grades of malnutrition and all died while under observation, either in the ward or out-patient department, from nutritional causes, None of them developed other evidences of tetany.

Although Escherich describes the test as easy to perform without resistance on the part of the patient, in my subjects there was constantly so much struggling that it was difficult to find the moment of muscular relaxation necessary for the appreciation of response to the weaker currents. In a small number of cases the reactions of both left and right sides were taken and very little difference found. Escherich spoke of considerable variation in his experience. Constant resistance necessitated mild chloroform narcosis in a few cases. The reactions obtained in this way were no lower than the average without such narcosis.

Escherich and von Pirquet considered the peroneal nerve muscle-group the best for use, and this proved the most satisfactory in my series. Erb employed the ulnar and prefers it. The median nerve has been used by Mann and Thiemich but reacts to anodal closure with less current than to kathodal closure, quite contrary to the findings in the peroneal nerve, and is in general more sensitive to electrical stimulation than the peroneal.

Muscular response was obtained at current strengths given in Table 1 in every one of the 318 tests made for kathodal closure. For anodal closure response occurred at 5 milliamperes, or lower, 149 times and failed 169 times. For anodal opening, eighty-seven results were positive and 231 negative. The reaction for kathodal opening was present only ten times and lacking in 308 instances. Table 1 shows the current strengths at which reactions were obtained.

Table 1.—Showing the Current Strengths at Which Electrical Reactions Were Obtained in Children.

	Kathodal Closure	Anodal Closure	Anodal Opening	Kathodal Opening
Less than 1 milliampere	0	0	0	0
From 1 to 2 milliamperes	37	2	0	1
From 2 to 3 milliamperes	74	28	3	3
From 3 to 4 milliamperes	58	53	23	2
From 4 to 5 milliamperes	29	19	17	3
From 5 to 10 milliamperes	18	57	47	30

The peroneal nerve-muscle group was employed for the tests, the negative electrode being placed on the upper abdomen, the positive over the peroneal nerve as it passes behind the head of the fibula.

The child to be tested was laid with the feet pointing to the left of the operator, whose left hand supported the left ankle and foot of the subject in such a manner as to feel any twitch occurring in the flexors of the ankle or of the toes. Slight reactions are readily felt in this way which would be imperceptible to the eye. The positive electrode is controlled by the operator's right hand, the negative one being held in position by the assistant who with his free hand controls the rheostat. It is necessary that the tests be begun with a current of sufficient strength to produce muscular response, and gradually be reduced from this to the point at which the twitch is lost. If the reverse is attempted the lowest contraction point will be invariably passed before response occurs.

Allowance for individual skin resistance is to be made in order to avoid undue disturbance to the patient through the use of unnecessarily strong currents. The resistance varies directly with the amount of subcutaneous fat, and reduces rapidly as the operation progresses, the latter fact being apparently due to the congestion which, developing under both electrodes, increases the skin's conductivity. This variation is great, both in different individuals and in the same case at the beginning and end of a test. Thus the same rheostat reading which in one instance may give a current through the body of 1 milliampere will in another case or after the skin congestion has developed during many applications of the current allow from 6 to 8 milliamperes to pass and result is unnecessary irritation and violent resistance on the part of the subject.

The test for kathodal closure should be first made, as response to this is obtained most readily and with the least current strength. Anodal closure, anodal opening and kathodal opening should follow in order.

Infants less than a month old are less susceptible to electrical stimulation than older children, and these in turn are more susceptible than the adult.

In general there are three grades of electrical irritability:

1. The normal, in which only kathodal closure occurs under 5 milliamperes. Sometimes anodal closure is found at 5 milliamperes or just below it.

2. The middle grade or anodal hyperirritability, in which kathodal closure is less than 5 milliamperes and anodal opening is less than anodal closure and less than 5 milliamperes.

3. Tetany, in which all four reactions are less than 5 milliamperes. As suggestive of tetany is the occurrence of anodal opening less than anodal closure and the appearance of kathodal-closure tetanus.

Of the children under discussion sixty-five were boys and fifty-three girls. In 24.6 per cent. of the boys and 15 per cent. of the girls hyperirritability occurred. Of the ten patients showing the highest grade of irritability there were seven boys and three girls. There were eighteen under 1 week of age, fourteen from 1 week to one month, eleven from 1 to 2 months, thirteen from 2 to 3 months, twenty from 3 to 5 months, and fourteen each from 5 to 7, 7 to 9 and 9 to 13 months. Of the eighteen children under 1 week of age, the middle-grade reaction was obtained in no case. Of the fourteen from 1 week to 1 month it was present in 7 per cent. Of the twenty-four from 1 to 3 months old 8 per cent. showed this partial reaction. From 5 to 8 months 50 per cent. of the twenty-four cases evidenced anodal irritability. Of eighteen children of 8 months and older, it was obtained in 22 per cent.

The averages obtained in children of from 3 to 8 months were consistently lower than those of any other period. Those from 1 to 3 months gave the next lowest.

Escherich's conclusions as to the reactions of normal children are:

1. The first weeks do not differ from the later weeks of life in electrical irritability.

2. In normally developing children only kathodal closure is less than 5 milliamperes.

3. Anodal closure at less than 5 milliamperes is seldom found in the peroneal nerve during the first year.

Table 2.——Showing Electrical Reactions According to Age.

	Average	Number of Tests
Under one week, 18 children—		
Kathodal Closure	3.6	25
Anodal Closure	4	5
Anodal Opening	4.9	4
Kathodal Opening	5+	all
One week to one month, 14 children—		
Kathodal Closure	4.5	14
Anodal Closure	6.9	10
Anodal Opening	6.3	3
Kathodal Opening	7	1
One month to 3 months, 24 children—		
Kathodal Closure	3	44
Anodal Closure	4.3	26
Anodal Opening	5.3	17
Kathodal Opening	4.5	2
Three months to 5 months, 20 children—		
Kathodal Closure	2.7	42
Anodal Closure	3.9	34
Anodal Opening	4.4	17
Kathodal Opening	1.3	1
Five months to 8 months, 24 children—		
Kathodal Closure	2.9	70
Anodal Closure	3.5	56
Anodal Opening	4	33
Kathodal Opening	3.4	4
Eight months and over, 18 children—		
Kathodal Closure	3.8	24
Anodal Closure	4.5	18
Anodal Opening	4.6	9
Kathodal Opening	5	1

4. Opening contractions are seldom found at less than 5 milliamperes.

My experience seems to indicate that children less than 1 week old are less sensitive to electrical stimulation than when older. Anodal closure was elicited 100 times at a point less than 5 milliamperes. Anodal opening was found fifty-three times, and kathodal opening ten times. This variance of these results with those of Escherich may be due to the fact that my children were, as before stated, far from normal, in that they were all in the ward because of nutritional disturbances. To this point reference will be made later.

There were in this series six infants weighing less than 5 pounds, seventy-one between 5 and 10 pounds, and twenty-three of 10 pounds and over.

The incidence of partial hyperirritability in children under 5 years was 16 per cent.; 5 to 10 pounds, 22 per cent.; 10 pounds and over, 30 per cent.

With reference to change in weight or, as this is its best index, the general condition of the subjects, it was found that of the sixty children losing weight, 25 per cent. showed partial hyperirritability; of the twelve with stationary weight,

16 per cent. showed partial hyperirritability; and of the twenty-seven gaining weight, 37 per cent. showed partial hyperirritability.

During the period of this work there were fifty-seven infants on skim-milk mixtures, twenty-one on condensed milk, twenty-three on buttermilk, eighteen on Finkelstein's "Eiweismilch," fourteen on whole milk mixtures and twenty-four were on the breast. As it has been shown that tetany occurred more frequently in artificially than in breast-fed children, comparisons between the reactions of these classes were made. In the breast-fed the average of kathodal closure, 3.2 in thirty-eight tests; anodal closure, 4 in twenty-four tests; anodal opening, 4 in seven tests; and kathodal opening, 2.4 in one test, were practically the same as those for artificial feeding, which were: kathodal closure, 2.8 in 172 tests; anodal closure, 3 in 123 tests; anodal opening, 4 in sixty tests; and kathodal opening, 4 in nine tests. These averages of the values found to obtain in the individual tests are given to show something of their individual variation. Taking the number of breast and artificially fed children who showed distinct hyperirritability, it appears that there was none of the ten former, but 24 per cent. of the 108 of the latter class. It must be added, however, that the cases were not exclusively on either type of nourishment for a great length of time before testing.

Referring again to nutritive condition, the reactions were classified as to good and poor gastric and intestinal digestion. There were thirty-eight in which the gastric digestion was good, fifty-eight in which it was poor. Twenty-seven children had satisfactory intestinal conditions and in seventy-seven the opposite was the case.

Again leaving the questionable average figures and dividing these classes of digestive condition into those showing hyperirritability and those reacting to kathodal closure alone it appears that of thirty-eight instances of good gastric digestion 30 per cent. showed hyperirritability; of fifty-eight instances of poor gastric digestion 30 per cent. showed hyperirritability; of twenty-seven instances of good intestinal digestion 33 per cent. showed hyperirritability; of twenty-seven instances of poor intestinal digestion 24 per cent. showed hyperirritability.

Reviewing the tabulations concerning size, gain or loss in weight, digestive condition and method of nourishment, it is apparent that they throw little if any light on the relation of these conditions to electrical irritability. The strong impression was gained, however, during the progress of the work, that the child's irritability varies directly with the general condition of nutrition, and that the perfectly developed, well-nourished subject gives response much less readily to galvanism than if under-fed and of low body weight.

It is evident that from this compilation nothing can be stated in respect to nutrition and electrical irritability, except that a considerable series of tests showed no influence of the one on the other.

Table 3.—Showing the Electrical Reactions Under Varying Conditions of Digestion and Nutrition.

	Average	Number of Tests
With good gastric digestion—		
Kathodal Closure	2.8	88
Anodal Closure	5.3	60
Anodal Opening	3	39
Kathodal Opening	3.6	4
With poor gastric digestion —		
Kathodal Closure	3	133
Anodal Closure	3.6	111
Anodal Opening	4.3	53
Kathodal Opening	3.6	5
With good intestinal digestion—		
Kathodal Closure	3	38
Anodal Closure	4.1	44
Anodal Opening	4.1	28
Kathodal Opening	4.2	1
With poor intestinal digestion—		
Kathodal Closure	2.2	140
Anodal Closure	3.6	111
Anodal Opening	4.6	50
Kathodal Opening	3.6	4

Table 4.—Showing Average of Reactions in Table 3, Grouping Gastric and Intestinal Together.

	Average	Number of Tests
With good gastric and intestinal digestion—		
Kathodal Closure	4.9	126
Anodal Closure	3.7	104
Anodal Opening	3.0	67
Kathodal Opening	3.9	5
With poor gastric and intestinal digestion—		
Kathodal Closure	2.6	273
Anodal Closure	3.6	222
Anodal Opening	4.4	103
Kathodal Opening	3.6	9

Summary.

1. Tetany is seen in about 2 per cent. of infants under 1 year.

2. An early and definite diagnosis depends on the presence of marked electrical hyperirritability.

3. Normal children react to kathodal closure alone at less than 5 milliamperes.

4. Many children not having tetany give low response to anodal closure and in fewer cases to anodal opening.

5. This partial or anodal grade of irritability may depend on factors entirely foreign to tetany for its causation or may mark the early stages of the disease.

6. Infants of less than 1 month respond less readily to galvanism than do other children.

7. Kathodal opening contraction may be absent in true tetanoid conditions.

8. The degree of hyperirritability may vary greatly in the same case from time to time.

9. No constant difference was found in the reactions obtained on left or right sides.

10. Anesthesia does not affect electrical irritability.

11. The peroneal group is best adapted for the test.

12. Three points in the technic are important:

(a) The test must start with a current strength able to produce muscular response and must be carried on with reducing current strength to the point at which muscular response fails.

(b) As the skin conductivity is constantly increasing care must be taken to avoid undue and strong stimulation as the test progresses.

(c) Kathodal closure, anodal closure, anodal opening and kathodal opening should be tried in the order of ease of eliciting and the amount of current strength required; that is, in the order named.

13. The highest incidence of tetany is during the early months of the year.

The Electrical Reactions in Parathyreopriva Dogs.

A series of experiments on normal dogs, and on those deprived of part or all of their parathyroid tissue, was made with the object of determining:

1. The electrical irritability of the normal dog.

2. The influence on this irritability of the removal of one or more of the parathyroid glands.

3. The interval elapsing before the electrical evidences of tetany appeared.

4. The amount of time by which these symptoms antedated the physical evidences of the disease.

5. Whether the remaining glands would show evidences of attempt at compensatory hypertrophy.

The galvanic reactions of these dogs, taken for several days before operation to establish the normal for each animal before testing for abnormal reactions, followed very closely the normal reactions of the children tested in the ward. The same nerve muscle-group as that in the case of the child was used, the peroneal, the negative electrode being placed on the abdomen. No difficulty was met in getting accurate readings so long as the skin at the site of contact of the electrodes was kept closely shaven. It was unnecessary to use an anesthetic for the tests, and in the few cases in which mild anesthesia was used, for reasons of comparison, no change in the response to the current was observed.

Following the method used by McCallum, and in the belief that in the short time that these animals lived after operation no influence due to hypothyroidism would cloud the findings, thyroid and parathyroid glands were removed together. On account of the length of time necessary for such observations, it was not possible to include in this paper the ultimate results on the electrical reactions of simple partial parathyroidectomy. This will be made the subject of a later report.

The average reaction to kathodal closure before operation was from 1 to 3 milliamperes; for anodal closure, 3 to 4 milliamperes; for anodal opening, 4 to over 5 milliamperes; for kathodal opening, always over 5 milliamperes.

In one of the animals tested, only one parathyroid was removed, and in five cases, two parathyroids, with one lobe of the thyroid, were taken out. The intention here was to determine the effect on the electrical irritability of the nervous system of a lesion in a part of the parathyroid tissue. The animal with a single parathyroid gland removed showed only partial electrical irritability for 236 hours after operation, but there was immediate hyperirritability after the removal of the remaining three glands.

Of the semi-parathyreopriva dogs all five showed an electrical hyperirritability toward anodal closure and anodal opening. The kathodal reactions showed change in only two instances (Dogs 6 and 7). In two cases kathodal tetanus was elicited after the first operation, although it had not been obtained before the parathyroids were partially removed. This hyperexcitability to anodal stimulation appeared in from one to three days after removal of the two glands, and corresponds to Escherich's mild "Anodische grad."

In no case was there any evidence of tetany other than the changed electrical irritability. Lesions of one or two parathyroid glands may, then, produce moderate increase of electrical excitability to the galvanic current, but do not at once result in general tetany. In two instances (Dogs 6 and 7) partial thyreo-parathyroidectomy was followed by the complete tetany reaction. These animals might have furnished examples of tetany due to partial removal of the parathyroid tissue had the second operations been longer deferred. In every case of partial removal of the glands there appeared, in varying degree, some tendency to anodal irritability. Two of McCallum's semi-parathyroidectomized dogs showed tetany in two days after the operation on the glands of one side.

In four dogs both lobes of the thyroid, with entire capsule and all four parathyroid glands, were removed at one operation. Electrical hyperirritability to all four types of current supervened in from five to forty-eight hours after operation, with one exception. The electrical tests showed tetany from a few hours to two days before any other symptoms of the condition appeared. One of these four dogs showed slight laryngospasm on the sixth day following operation, but up to the fourteenth day gave no typical increased response to electrical stimulation. He was then given 2.5 gr. of thyroid extract daily to counteract any effect of the loss of the thyroid secretion, and did for four days respond to currents of lesser strength. This, however, did not persist, and the animal died on the forty-sixth day after operation without having shown any ill effect from the removal of all his parathyroid tissue. A careful autopsy failed to show any accessory glands in the neck, the thymus or elsewhere, or to furnish any additional clue as to the cause of the sudden death of the animal.

After the removal of all four glands, nine animals gave low electrical reactions, from which a diagnosis of tetany could be made in twenty-seven, twenty-one, twenty-two, sixteen, twenty-nine, nineteen, seventeen, nineteen, nineteen hours after removal. In two instances (Dog 3) kathodal closure, anodal closure, anodal opening, dropped to 4.0 miliamperes in twenty-six hours after operation and the animal died in tetany, but kathodal opening remained over 5 milliamperes.

Partial thyreo-parathyroidectomy was done on six dogs and the operation made complete, respectively, in four, five, twelve, twelve, thirteen and thirteen days later.

Measurements of the lobes of the thyroid and of the parathyroid glands first removed were compared with those left till the second operation. Search was also made in sections from all glands for mitosis and evidences of glandular over-activity. In each case there was evident hypertrophy of the remaining lobe of the thyroid, but there was found no evidences of attempt at similar change in the remaining parathyroid glands.

The accompanying charts indicate that the electrical reaction of animals subjected to parathyroid injury of varying degree do not change pari passu with the lesion produced, although Biedl and others have found that the general picture of tetany depends for its intensity on the number of glands removed. It was found, in the majority of cases, that after partial operation the reactions were only in small degree changed till the remaining glands were removed, when sudden tetany developed.

As it has been observed by several experimenters that parathyroidectomized animals often do not show symptoms of tetany till subjected to some metabolic or physical strain, electrical tests on the partially parathyroidectomized dogs were made after considerable muscular exertion. The nervous excitability was not found increased by such exercise, although tachypnea, muscular spasm, and laryngospasm were frequently induced or augmented. The response to galvanism is, then, less influenced by intercurrent conditions than other tetany symptoms.

Electrical hyperirritability resulting on removal of all parathyroids appeared in nine of the ten dogs tested, sixteen, six, twenty-four, and twenty hours and one, one, two, three, and four and one-half days, respectively, before the development of other tetanoid signs.

The average time of onset, after complete operation, of electrical hyper-

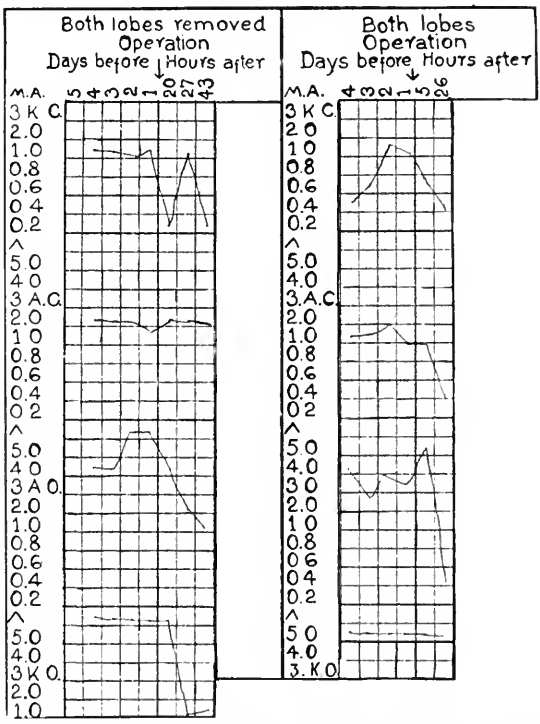

Chart 1, Dog 1.—Electrical reactions after complete extirpation of glands, weight of dog 22 pounds; killed forty-eight hours after operation; no signs of frank tetany. Electrical hyperirritability preceded death sixteen hours.

Chart 2, Dog 2.—Weight 27.5 pounds; died in tetany thirty-one hours after operation. Electrical irritability preceded tetany six hours.

irritability, was thirty-three hours. The average for the same in cases in which the operation was done in two stages was twenty hours. Inasmuch as in the short time allowed to elapse between operations in this series of experiments there seems to have been produced a disposition to immediate response to further injury, it is possible that more time would have allowed the development of tetany reactions after only partial extirpation of the parathyroids.

Symptoms referable to the hyperirritability of nerve centers were, then, in every instance, the first to appear, and although in this series of tests they preceded other symptoms by an apparently short time, it is to be remembered that in no case was the course of the disease produced longer than sixteen days,

the average being nine days. The average of the times occupied in the development of electrical hyperirritability was one-ninth of the times lapsing before the production of other symptoms.

The galvanic current is, then, of especial value in the early diagnosis of tetany.

Analysis of Parathyreopriva Dogs

A. Complete Extirpation of Glands.

Dog 1.—Female, weight 22 pounds. August 11, reactions were obtained at kathodal closure, 1; anodal closure, 2; anodal opening, 3; kathodal opening, over 5 milliamperes. Under ether anesthesia anodal tetanus was obtained at anodal opening, 3 milliamperes; the other reactions being unchanged.

Augst 12, closing contractions somewhat lower, opening contractions over 5 milliamperes. No change with ether anesthesia.

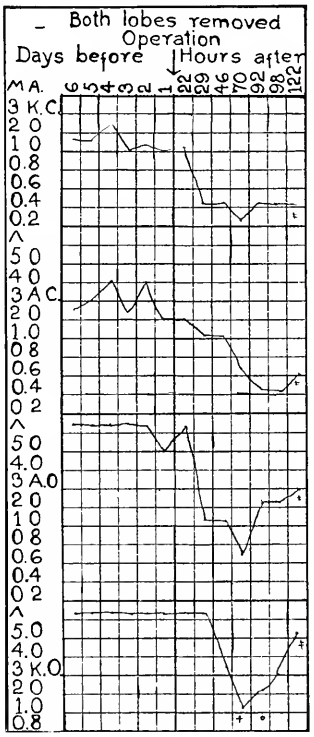

Chart 3, Dog 3.—In the above chart "o" = no general tetany; + = general tetany; ± = tests made as dog was dying, showing further reduced reaction to opening contractions.

August 15 and 16, reactions the same. The average for this animal's galvanic response was kathodal closure, 1; anodal closure, 2; anodal opening, 4; kathodal opening over 5 milliamperes.

August 16, both lobes of thyroid with capsule were removed and found to include four parathyroid glands all lying on surface of thyroid.

August 17, twenty hours after operation, there was no evident tetany. The reactions were, kathodal closure, 2; anodal closure, 2; anodal opening, 4; kathodal opening over 5 milliamperes. Twenty-seven hours after operation there was further evidence of electrical hyperirritability, reactions being obtained at kathodal closure, 1; anodal closure, 2; anodal opening, 1; kathodal opening, 1.1.

August 18, forty-three hours after operation, there was fine tremor, slight rigidity and tachypnea, with reactions practically the same as those of the preceding day. On account of considerable distress the dog was killed two days after operation.

Dog 1 showed electrical hyperirritability 20 hours after operation and one day before there was other evidence of tetany.

Dog 2.—Male, weight 27 pounds. The average of the reactions taken on each of the four days before operation was, kathodal closure, 1; anodal closure, 2.4; anodal opening, 3.6; kathodal opening over 5 milliamperes.

August 18, complete thyreo-parathyroidectomy was done, four glands being identified. Five hours after operation the reactions were, kathodal closure, 3.

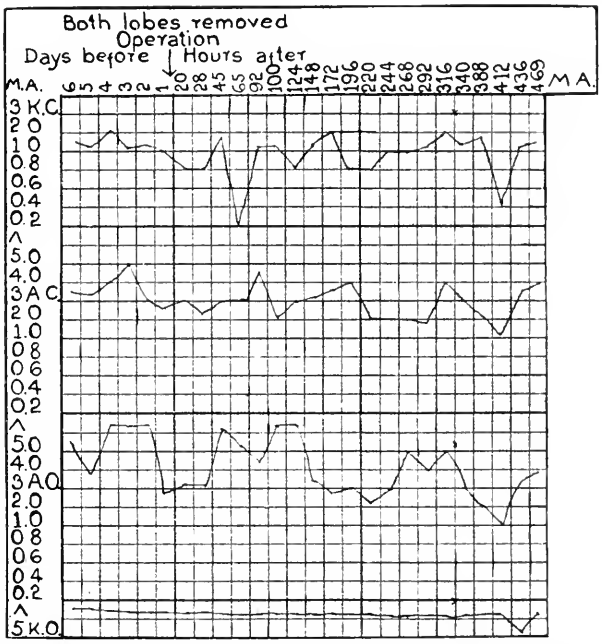

Chart 4, Dog 4.—In the above chart the cross (x) indicates that 2.5 gr. thyroid extract was given daily; dog weighed 6 pounds; died forty-six days after operation suddenly, having had no signs of tetany other than laryngospasm.

anodal closure, 2; anodal opening, 5; kathodal opening over 5 milliamperes. Twenty-three hours after operation the reactions were, kathodal closure, 2; anodal closure, 2; anodal opening, 2; kathodal opening, over 5 milliamperes. Twenty-seven hours after operation muscular twitchings were noticed and the animal died in convulsions and general tetany four hours later.

This dog showed an unusually prompt onset of electrical hyperirritability and frank tetany with death in severe muscular spasm in thirty-one hours after operation. With the exception of kathodal opening, the reactions were very low, but the former never dropped below 5 milliamperes, although the physical evidences of tetany were severe.

Dog. 3.—Six observations during the week preceding operation showed the normal reactions to be, kathodal closure, 1.3; anodal closure, 2.4; anodal opening and kathodal opening over 5 milliamperes.

August 23, both lobes of thyroid with four parathyroid glands were removed.

August 24, twenty-two hours later, there was increased excitability to clos-

ing, but none to opening tests. Twenty-nine hours after operation, however, all tests responded to currents of less than 1 milliampere, excepting kathodal opening, which remained above 5 milliamperes.

August 25, forty-six hours after operation, kathodal opening dropped to 3 milliamperes, and seventy hours after operation, all reactions were below 1 milliampere. From this time on to the death of the dog the reactions remained

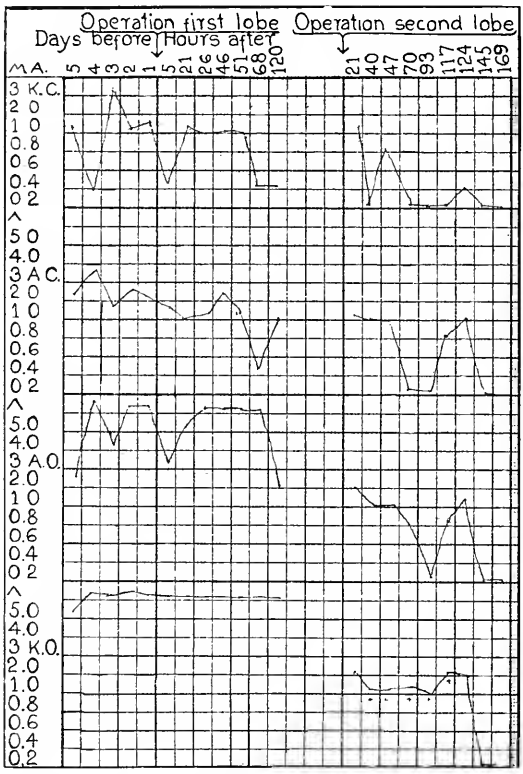

Chart 5, Dog5.—Weight 32 pounds. x = occurrence of tetany (kathodal); first evidence of frank tetany four days after second operation; dog killed 169 hours post operatione.

about the same. Tests made at a few moments previous to death gave still further reduced reaction to opening contractions.

Frank tetany appeared forty hours after operation and persisted throughout. In this case the electrical indications of tetany preceded only by a few hours the physical symptoms of the disease.

This dog evidenced early and marked response to parathyroid lesion by electrical hyperirritability and only five hours later all tetany symptoms were pronounced.

Dog 4.—Female, weight 6 pounds. Daily observations for six days preceding operation gave this animal a normal average of kathodal closure, 1.2; anodal closure, 3; anodal opening, 5; kathodal opening over 5 milliamperes.

August 24, complete thyreo-parathyroidectomy was performed and for six days no change was noticed other than slight increased responsiveness to kathodal closure and anodal closure. On the sixth day moderate laryngospasm developed and this persisted as the only symptom of tetany till death, which occurred

on the forty-sixth day after operation. Autopsy failed to show the existence of accessory parathyroids. Fifteen days after operation 2½ gr. of thyroid extract was given daily to counteract any effect of the loss of the thyroid lobes. This had no effect on the general condition.

This animal alone failed to develop hyperirritability or marked tetany symptoms after suffering loss of all the parathyroid glands.

B. Dogs in Which Parathyroidectomy Was Done in Two Stages.

Dog 5.—Male, weight 32 pounds. For the six days preceding operation the average reactions were found to be, kathodal closure, 1; anodal closure, 3;

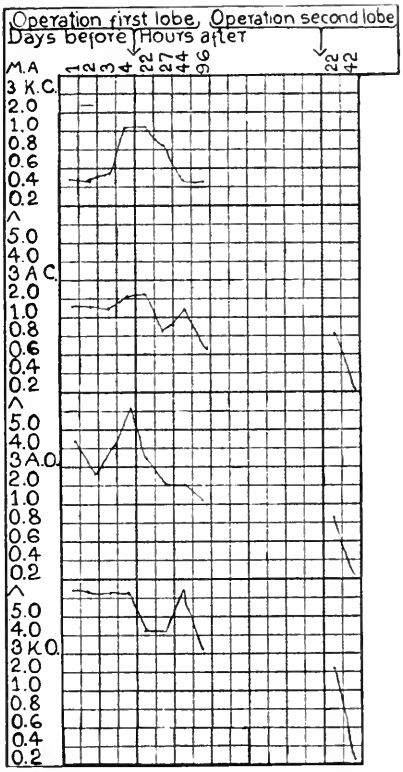

Chart 6, Dog 6.—Weight 30 pounds. Killed in tetany forty-eight hours after second operation. Electrical hyperirritability preceded tetany forty-four hours.

anodal opening and kathodal opening over 5 milliamperes; i. e., relatively low closing contractures.

August 17, the right lobe of the thyroid with capsule was removed, two parathyroid glands being identified. Through the slipping of a ligature there was severe hemorrhage, without, however, any ill effects being manifest, then or later. There was a gradual rise in electrical excitability for kathodal closure, anodal closure, and anodal opening for five days. At the end of this time the figures being, respectively, 0.4, 1, and 2 milliamperes. There were no other signs of the tetany condition. On August 22, the remaining lobe of thyroid, which was markedly hypertrophied and contained one parathyroid, was removed. Again, such severe hemorrhage occurred as to make recovery doubtful.

Twenty-one hours after the second operation the electrical diagnosis of tetany was definite, with kathodal closure, 1.4; anodal closure, 1.1; anodal opening, 2.1; kathodal opening, 2.1 milliamperes. The reaction points dropped steadily until August 20, when all were obtained at .2 milliamperes.

No physical evidences of tetany were noted till August 26, when there was some tachypnea. On August 29, the dog was stupid, emaciated and would not eat, but had no definite tetany symptoms. He was chloroformed at this time.

For four days preceding the second operation this dog was evincing an increasing electrical hyperirritability which continued uninterruptedly after the

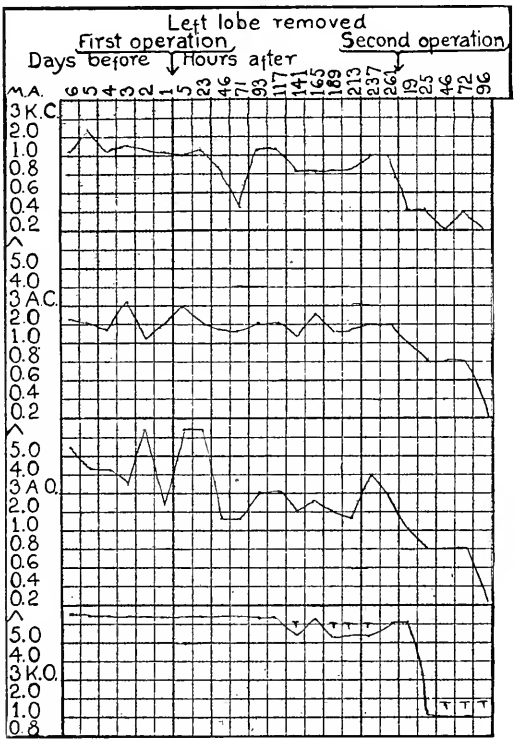

Chart 7, Dog 7.—Died in tetany ninety-six hours after second operation. "T" = kathodal tetanus.

second operation. Although the electrical reactions had been strong for tetany, for nine days there was no other evidence of the condition, until August 26, when mild tachypnea developed. McCallum has shown that the muscular spasm, convulsions, etc., of experimental tetany can be controlled and temporarily relieved by bleeding. The second severe hemorrhage may in this case explain the absence of frank tetany. If this supposition be granted, it is of interest and consequence to note that the loss of blood exerted no influence on the course or the development of the nervous hyperexcitability.

Dog 6.—Female, weight 30 pounds. The main reaction before operation was found to be kathodal closure, 1.4; anodal closure, 2; anodal opening, 4; kathodal opening over 5 milliamperes. On August 18, the thyroid and parathyroids of the left side were removed. During the following four days there was distinct increase of the electrical hyperirritability, the reactions being, kathodal closure,

.4; anodal closure, .6; anodal opening, 1; kathodal opening, 5 milliamperes. This increased irritability began twenty-seven hours after operation. August 22, the remaining lobe of the thyroid, with its parathyroids, was removed and, at once, all reactions dropped to .2 milliamperes. No other signs of tetany were present till two days after the second operation.

This dog responded markedly in her nervous excitability to the removal of part of her parathyroid tissue, and gave low reactions for four days before mus-

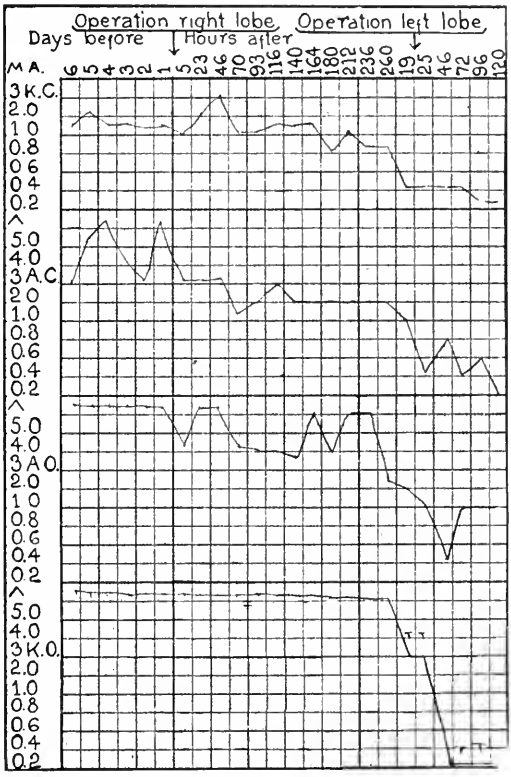

Chart 8, Dog 8.—Weight 16 pounds; found dead September 12, 120 hours after second operation, never having shown signs of apparent tetany.

cular spasm appeared. Moderate hypertrophy of the remaining thyroid lobe was noted, but no change was apparent in the parathyroids.

Dog 7.—Male, weight 11 pounds. Six tests gave for this animal kathodal closure, 2; anodal closure, 2; anodal opening, 5; kathodal opening over 5 milliamperes as the average normal.

August 24, the left lobe of the thyroid and only one parathyroid were removed. During the ten days following operation there was constantly increasing irritability, most marked in the low anodal opening contraction and kathodal opening tetanus. No other tetany symptoms were noticed. September 6 the right lobe of the thyroid with remaining parathyroids was removed. All reactions fell to 1 milliampere or lower, twenty-three hours after the second operation. Three days later muscular spasm was severe and the dog died in tetany.

Dog 8.—Female, weight 16 pounds. This dog, reacting normally for six days at kathodal closure, 2; anodal closure, 5; anodal opening and kathodal

opening, over 5 milliamperes, was deprived of left lobe of thyroid and two parathyroids on August 24.

No electrical hyperirritability and no signs of tetany were noted up to September 6, when the remaining glands were removed. Nineteen hours later the reactions were, kathodal closure, .4; anodal closure, 1; anodal opening, 2; kathodal opening, 3 milliamperes. This hyperexcitability steadily increased till

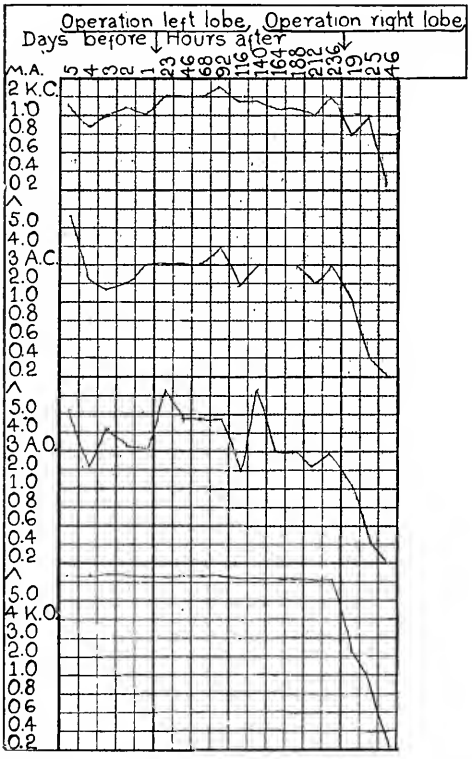

Chart 9, Dog 9.—Weight 21 pounds. One parathyroid removed at first operation; frank tetany appeared forty hours after second operation, with paralysis of front legs; dog killed forty-six hours after second operation.

death occurred suddenly five days after operation, there having been up to this time no other evidences of tetany.

There was in this case also hypertrophy of the second lobe of the thyroid, but none apparent in the parathyroids.

Dog 9.—Male, weight 21 pounds. The reactions of this dog before operation were, kathodal closure, 1; anodal closure, 3; anodal opening, 4; kathodal opening over 5 milliamperes. After removal of left lobe of thyroid, in which only one parathyroid was found, there was no increase in sensitiveness to galvanism or muscular spasm for nine days, at the end of which time the right thyroid lobe, with two parathyroids, was excised. Nineteen hours later the reactions were, kathodal closure, .8; anodal closure, 1; anodal opening, 1; kathodal opening, 2 milliamperes. Forty hours after operation there was partial paralysis of the front legs, with electrical reaction, all below .2 milliamperes.

Here, again, injury to a portion of the parathyroid glands caused none of

the phenomena of tetany, but apparently prepared the way for a very rapid and severe reaction on the ablation of the remaining glands.

Dog 10.—Female, weight 18 pounds. This dog's reactions before operation were; kathodal closure, 1; anodal closure, 3; anodal opening, 5; kathodal opening over 5 milliamperes. After removal of the left half of the thyroid and parathyroid tissue, the reactions of kathodal closure, anodal closure, and anodal opening were somewhat irregular, with kathodal opening unaffected, until the extirpation of the remaining glands, nine days later. Nineteen hours after second operation there was marked hyperirritability, and a day and a half later electrical

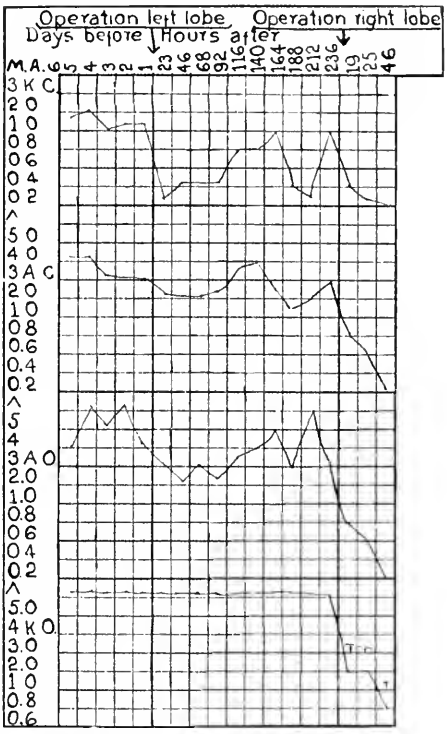

Chart 10, Dog 10.—Weight 18 pounds; frank tetany came on seventy hours (killed) after second operation.

reactions were obtained below .8 milliamperes, and there was severe general tetany.

Summary.

1. The reactions in normal dogs are similar to those of normal children.
2. The peroneal nerve muscle-group is best suited to the test.
3. Mild anesthesia does not increase the delicacy of the test.
4. Both sides of the body react alike.
5. In normal dogs kathodal closure averages at from 1 to 3 milliamperes; anodal closure from 3 to 4; anodal opening from 5 to over 5, and kathodal opening is always over 5 milliamperes.
6. The complete extirpation of the parathyroid glands produces prompt hyperirritability.
7. Partial extirpation of the parathyroid glands produces either anodal irritability or only inconstant change.

8. Complete extirpation, following partial removal of the glands, results in more immediate hyperirritability than when all glands are removed at once.

9. Increased irritability follows the complete parathyroid extirpation in an average of thirty-three hours.

10. Electrical hyperirritability follows the second of the two-stage operation in an average of twenty hours.

11. Electrical change always precedes the other symptoms of tetany by considerable time.

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THE BLOOD PRESSURE INDEX OF ECLAMPSIA.*

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The greatest danger confronting the pregnant woman is eclampsia. Concerning its origin and treatment little progress has been made and the death rate is still in the neighborhood of twenty-five per cent.

Suddenness of onset in an apparently healthy woman is one of the characteristics of the disease and from time to time various clinical signs or symptoms have been heralded as indices to the pre-eclamptic condition. In about one-fourth of the cases there are present certain premonitory subjective signs which are indisputable, but the convulsions may follow in a few hours so that there is hardly time to adopt any preventive régime.

For many years changes in the urine have been considered the earliest premonitory signs but the value of these changes have been greatly overestimated. Albumin and casts are almost always present in the pre-eclamptic stage and in the actual condition they are usually present in considerable amount and numbers, but numerous attacks occur while the albumin exists only as a trace, in fact scarcely more than might be considered the normal or physiological albuminuria of pregnancy. Indeed it is probable that the disease is an autotoxic condition produced or accompanied by degeneration of the liver cells and that the kidney lesion is only a secondary one. Seventy-five per cent. of the cases show no abnormal urinary signs a short period after delivery (1).

Percentage urea as determined by the common clinical tests is of no value. When the amount of urine passed during twenty-four hours can be measured and the total nitrogen and the urea nitrogen can be determined by chemical laboratory methods, distinct value may be assured. Further, if any definite value is to be given this index it must be determined every two or three days in the last month of pregnancy instead of weekly or fortnightly as the general custom is at present. The time necessary for such an elaborate series of tests would place the cost of this form of insurance so high that it would not be acceptable even to the very wealthy.

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A number of times the attention of the profession has been called to the value of blood pressure as an index to the onset of the eclamptic condition. Much credit is due to Janeway for the clear way he has presented this subject in his book on blood pressure. To determine the value of this index several factors must be considered.

First. What is the average blood pressure of the normal woman in the latter months of pregnancy, and what are the causes of variations from this average?

Since October, 1909, it has been the custom on Dr. J. C. Edgar's Bellevue

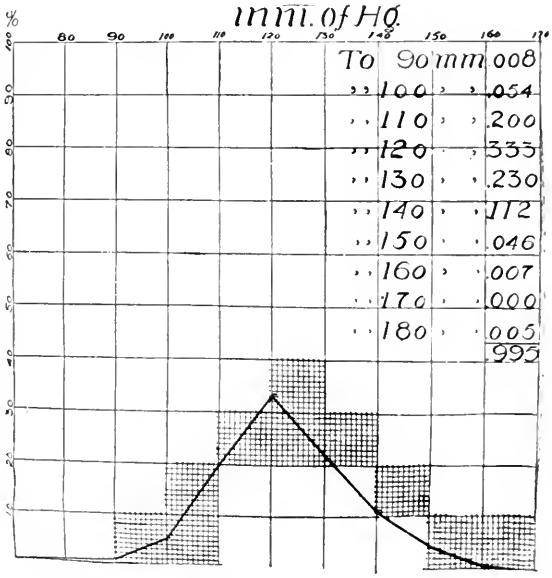

Chart 1.—Range of blood pressure in 1,131 readings on 145 women.

service to make frequent blood pressure readings on the waiting women. Most of these examinations have been made therefore in the last four or five weeks of pregnancy.

In 1,136 systolic readings on 145 women it has been found that the average blood pressure is 118 mm. of Hg. These readings were made with the wide cuff Stanton instrument and do not include readings taken on the day of labor. J. C. Hirst (2) also found the average to be in this neighborhood. To state an average in this condition gives a very inadequate picture, for many women have decided variations in the pressure taken at different intervals. I have made a diagram showing quite accurately the range of pressure in these 1,136 readings.

It will be readily seen that these readings vary greatly but that there is a high limit rarely passed. This is quite in accord with Janeway's statement (3) that a tension above 160 mm. does not exist in normal pregnancy. Some women run a continuously low blood pressure without apparent cause. Chart II shows the readings of a perfectly healthy multipara.

Many women run a continuously moderate blood pressure which is little affected by the ordinary occurrences of ward life, but by far the most numerous are those having from day to day an irregular range of blood pressure.

As far as it could be determined there was no regular variation in the read-

ings from primiparae and multiparae. Of these women 28 per cent. showed a variation of 25 to 30 mm. during the course of some days.

A study of the nitrogen partition of the urine of the women who have marked changes from day to day has so far been found to be of little value, but this work is being continued with the hope that slight change of metabolism may be coincident with increased pressure and thus a control be found for this index.

Observations through the remainder of the pregnancy in this type of case lead to the conclusion that changes of 30 mm. above the normal average (118) mean very little from a practical standpoint. Excitement, exertion, digestion,

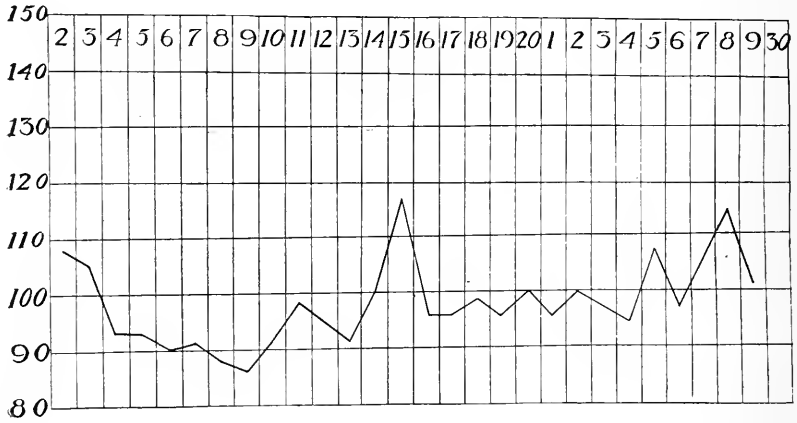

Chart II.—Low range of blood pressure, last month of pregnancy, June, 1910.

rest, and sudden changes of position all tend to produce changes up to 10 mm. but seldom more than this amount. Chart IV taken from Crile's book on blood pressure shows physiological variations according to Weiss (4). His estimations were made with the tonometer.

The onset of labor tends to raise the blood pressure. In the first and second stages of labor 140—150 mm. may be considered a fair average, if taken between the pains (5).

Second. Is the blood pressure invariably high in eclampsia and at about what point may it be considered as abnormal or as evidence of the pre-eclamptic state?

Eclampsia is a toxaemia or autotoxaemia which has for its most prominent or striking symptom the convulsive seizure. The toxæmia of the early months of pregnancy is often followed by the eclamptic condition in the latter months and it has been said with reason that the eclampsia is only a further product or advanced condition of the autotoxic state occurring in the early months (6).

Examinations of blood pressure in early toxæmia in our own cases and the cases of others were invariably low. Apparently toxic substances are circulating in the blood which have marked influence on the vomiting center, but with little action on the vasomotor apparatus either central or peripheral.

In the developed toxæmia of the latter months there is usually present a blood pressure raising principle or else by hormone action or similar means the pressure is raised to increase the natural resistance of the body. The fact that in the ful-

minant type of fatal toxæmia in the latter months the blood pressure is very low points more to the latter idea and also tends to more closely associate the early and late manifestations of poisoning that occur in these women.

To show how frequent these fulminant types are a description of two cases will be given that occurred on the Bellevue service during the past year.

Case 1. K. F., para five, about 7½ months pregnant was admitted to Dr. Edgar's service at Bellevue, April 8, 1910. She was brought in as a case of alcoholism, having been found in a stuporous condition in one of the city parks.

On admission she was semiconscious and cyanotic. She frequently vomited large quantities of black watery fluid which on examination was found to contain blood.

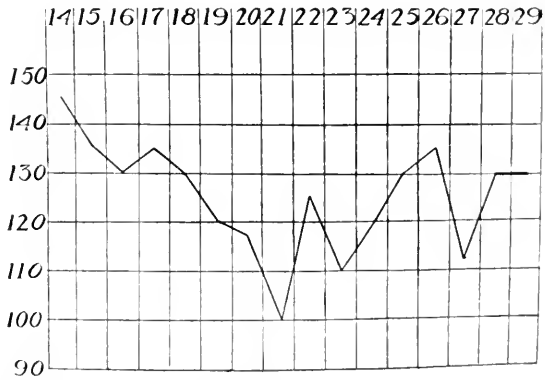

Chart III.—Irregular range of blood pressure, last month of pregnancy, June, 1910.

Her general condition was poor, the blood pressure was 70, and the pulse could barely be felt at the wrist. The urine, which black, was found to contain a large amount of albumin and hyalin, granular and epithelial casts. No fœtal heart sounds were heard. Her stomach was washed and she received caffeine, strychnine, hypodermoclysis and high colonic irrigation. Four hours after admission a hydrostatic bag was inserted into the uterus without anesthesia and ten hours later she expelled a dead 7—8 months fœtus, the placenta coming away shortly afterward. Blood pressure just before delivery was still 70 mm. Catheterization at intervals obtained in all but 16 ounces of urine. The morning of the following day she appeared somewhat better, was excreting more urine and the blood pressure was 80 mm. She died 33 hours after admission.

Case 2. M. S., multipara, seven months pregnant, was admitted to Dr. Edgar's service at Bellevue Hospital, October 31, 1910. She had been several days in a police station charged with intoxication. On admission she was stuporous, irrational, and unable to give any history. She had dyspnœa, air hunger, and a faint radial pulsation. The heart was irregular and the blood pressure could not be obtained. Her urine was loaded with albumin and casts and was dark in color. She received stimulation and saline intravenous infusion. On November 1st, she was a little better and her blood pressure was 75 mm. A 7 months fœtus was protruding into the vagina and was removed together with the placenta. She continued to improve, the blood pressure on November 3d reaching 80 mm. but the urine was scant in quantity (eleven ounces on November 1st, 13 oz. on November 2d) and still contained albumin, blood, and casts. She vomited frequently. On

November 4th, or four days after admission, she developed a pneumonic process in the right upper lobe and died within twelve hours.

While these two cases may be considered unusual the point to be emphasized is that a severe toxæmia may exist without convulsions and without high blood pressure, but in every other way similar to eclampsia.

While it is generally known that cases of eclampsia at the time of the convulsions usually have high blood pressures, still convulsions occur when the pressure

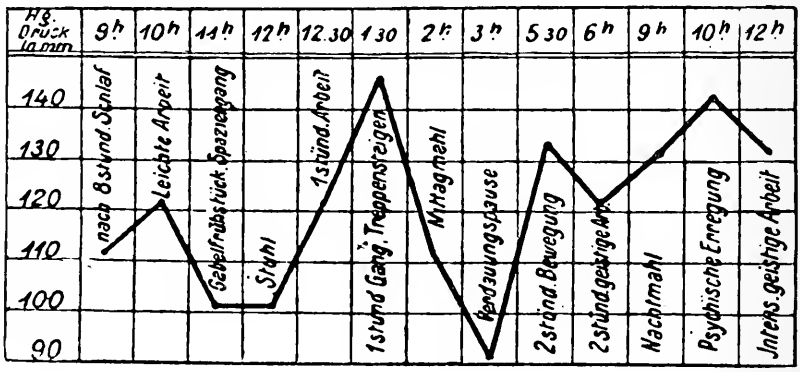

Chart IV.—Physiological variations in the blood pressue (Weiss.)

is as low as 155. Chart V shows the blood pressure readings of a typical case where the pressure was but moderately high.

Vogeler (7) and also Green (8) have attempted to classify the cases where the pressure was above the normal. In Vogeler's classification the high pressures are separated into three groups:

1st: Those moderately elevated and due to acute or chronic nephritis, 2d, those due to arteriosclerosis and 3d, those due to eclampsia.

This classification or any classification not taking into account the individual can hardly hold good. While it is true that the blood pressure is raised in both chronic parenchymatous and chronic interstitial nephritis, it would appear that the height of the pressure would be controlled by the coincident cardiac hypertrophy and vessel sclerosis.

Marked arteriosclerosis of itself does not occur at the age at which eclampsia is common.

It is evident then that convulsions in eclampsia though usually occurring at a time when the blood pressure is in the neighborhood of 200 may occur when the pressure is as low as 155, and furthermore that the eclamptic toxæmia may be even more severe when the pressure is very low. From the analysis of blood pressure readings by many observers 150 mm. may be taken as a danger limit and any pressure above this should demand rigorous investigation and treatment. Special mention should be made of Vogeler's conclusions to the same effect.

Third. What treatment should be adopted if the blood pressure index is accepted as indicating toxæmia?

The treatment should be entirely concerned with eliminating and limiting the production of the poisons. This may often be accomplished by active catharsis, sweating, diuresis, and limiting the nitrogen intake by placing the patient on a low calorie milk diet.

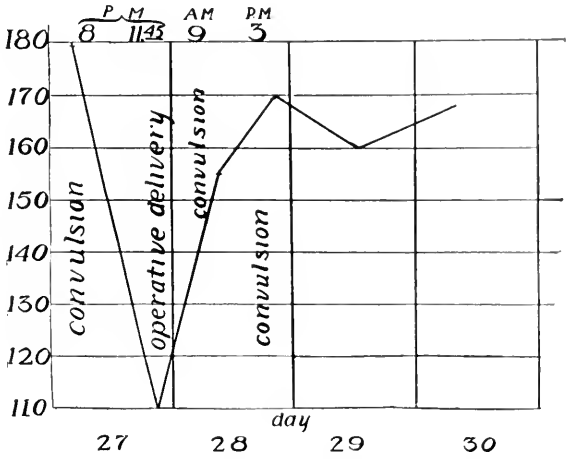

Chart V.—Eclampsia convulsions occurring at moderate blood pressure, November, 1910.

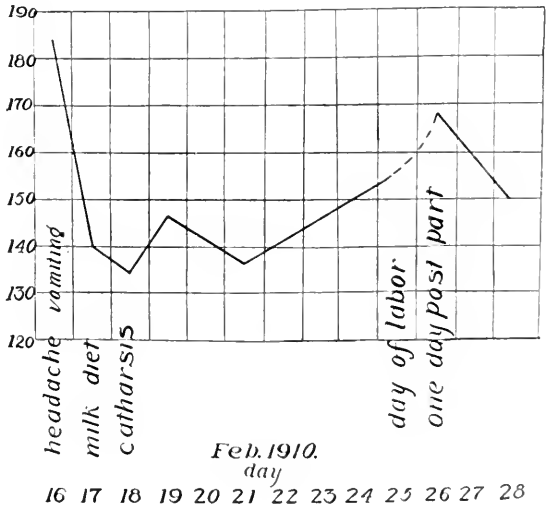

Chart VI.—Pre-eclampsia; rest, catharsis, and milk diet.

With the elimination of the toxins the blood pressure falls and this fall may possibly be an indication of the extent to which the treatment should be pushed. Even if convulsions ensue there is no reason to direct efforts to the lowering of the blood pressure, for the chronic nephritic and the arterio-sclerotic individual goes

about with a relative amount of safety with a blood pressure of 200 mm. There is every reason to suppose that the resilient arteries of these comparatively young women are equally able to withstand such a blood pressure.

If vessel dilatation is adopted by action on the medullary centers of such a drug as veratrum viride true collapse is produced (9). This effectually stops the convulsions it is true, but it leaves the patient in a condition similar to those cases of the fulminant type of the disease cited in this paper.

If vessel dilatation is adopted by means of such drugs as nitroglycerin and erythrol tetranitrate, which act for the most part at least on the peripheral mechanism

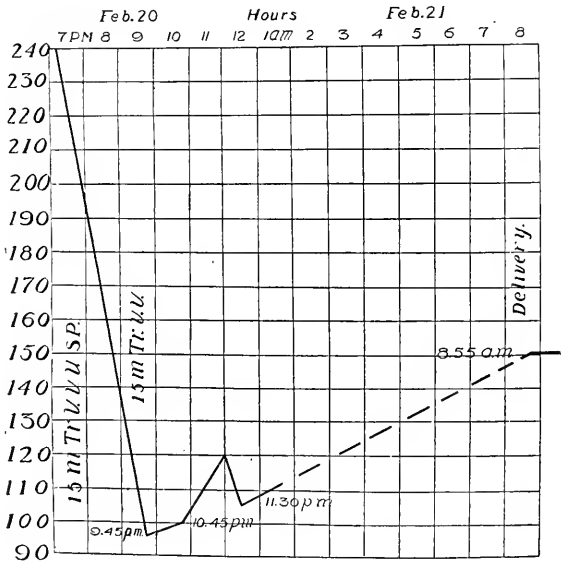

Chart VII.—Blood pressure. Pre-eclamptic state; veratrum viride action, fall of 145 mm. Hg. within three hours.

of the blood-vessels, shock or collapse does not occur, but the natural resistance of the organism toward the poison is reduced and the index to the success of the eliminative treatment is destroyed. I am not entirely convinced that when the pressure is over 200 mm. it may not be wise to reduce it to approximately that limit. It is well to remember the work of Wallace and Ringer (10), which shows that with high pressures the nitrite group is almost invariably successful in accomplishing an 11 to 14 per cent. reduction of the pressure.

The history of the disease shows that eclampsia rarely occurs as late as five days after delivery and that postpartum eclampsia is never as fatal as the antipartum or intrapartum conditions. The logical deduction is that the uterus should be emptied in all cases resistant to eliminative treatment where the blood pressure is high and is increasing.

Triweekly blood pressure examinations in the last four weeks of gestation will

give the closest clue to the pre-eclamptic condition obtainable at present and while not infallible offers combined with the ordinary methods now employed, the best index.

CONCLUSIONS.

1. Average blood pressure in the last weeks of pregnacy is 118 mm. of Hg. Fluctuations amounting to 30 mm. of Hg. above this need cause no alarm.

2. Blood pressure over 150 should be thoroughly investigated at once.

3. Blood pressure in eclampsia with convulsions, though usually in the neighborhood of 200 mm of Hg., may be as low as 155 mm.

4. Convulsions do not occur when the blood pressure is lowered by poor resistance, as in the so-called fulminant cases or when lowered by veratrum viride or other drugs producing collapse.

5. Treatment should be directed not toward reducing the blood pressure, but to the treatment of the toxaemia for the rise of blood pressure may denote only the resistance of the system toward the toxins.

6. Triweekly blood pressure examinations combined with the regular urine examinations for albumin and casts offer the best safeguard against the unexpected presence of this disease.

I am indebted to my friend Mr. Frederick Wengenroth for the drawing of the charts.

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SHOCK IN ECLAMPSIA.*

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At present there is a distinct tendency on the part of the profession to desert the radical obstetrical treatment of eclampsia and to return to the medical treatment, relying on veratrum for the control of the convulsions and stimulation of the skin, kidneys and bowels for the elimination of the poison.

In this country Hirst (1) and Zinke (2), both men of large experience, have come to the conclusion, from the study of their mortality lists, that medical treatment offers the best prognosis.

The object of this paper is to discuss certain observations made on eclamptic and pre-eclamptic cases during the past year, with special regard to the shock attendant

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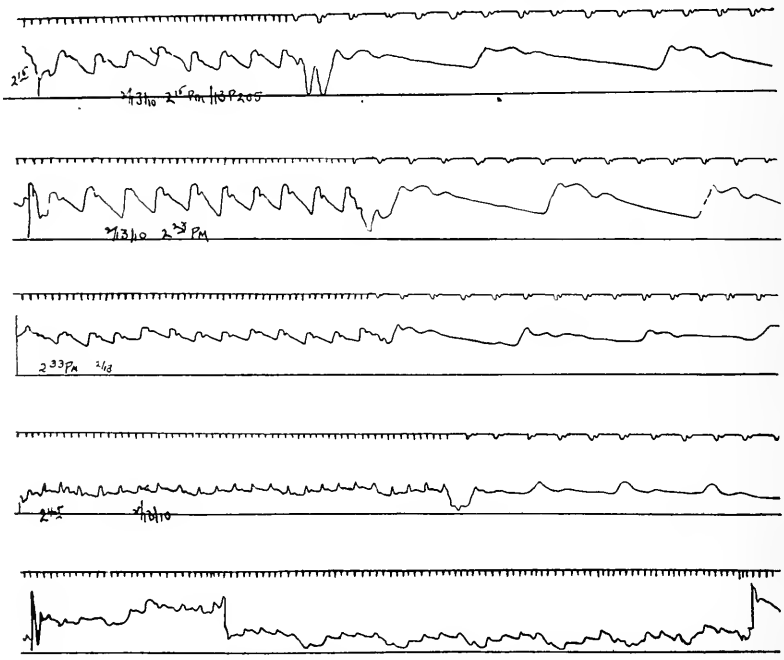
upon delivery and the shock caused by drugs used for the rapid reduction of blood pressure.

Shock may be described as partial or complete loss of central vaso-motor control due to profound injury to the tissue, loss of blood, or to severe heart depression. This definition would assume that collapse and shock are of the same nature, differing only in degree. Cook and Briggs and Crile believe that the earliest manifestation of shock and collapse is a fall in blood pressure.

Examination conducted by us and by others show that there is a rise of blood pressure during labor. The normal blood pressure during the first and second stages of labor may be placed at 130 to 150, if taken between uterine contractions (3). Following delivery there is a fall from 10 to 30 mm. of Hg. or practically a return to normal. This fall may be interpreted as being due to relief of pain and excitement



TRACINGS OF RADIAL PULSE.

1.—Before medication or delivery. Blood pressure 205 mm.
2.—At time of extraction of child.
3.—After 20 m. Tinct. veratrum viride and three minutes after completion of third stage.
4.—Fifteen minutes after completion of labor.
5.—Forty-five minutes after labor. Combined effect of shock and veratrum. Blood pressure 104 mm.—a drop of 101 mm. in one hour.

and possibly to some dilatation of the splanchnic area following the diminution of the size of the uterus.

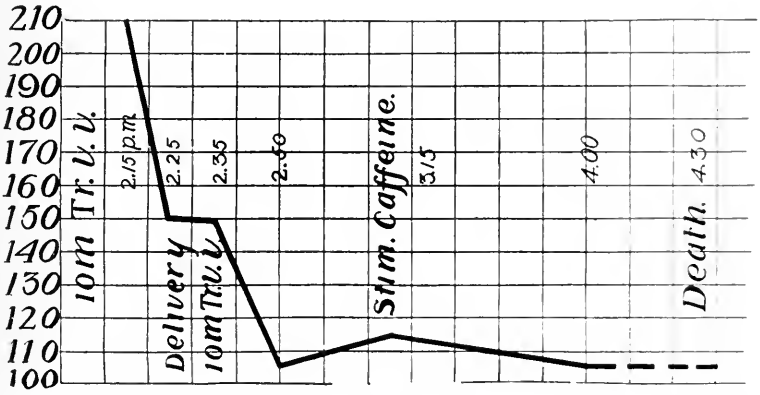
In eclampsia the blood pressure is inavriably high at the onset (excluding certain cases of grave toxemia occurring without convulsions in the latter months of pregnancy) (4). A fair average of the blood pressure readings at the time of the convulsions would be 200, although in one of the cases to be reported a convulsion occurred when the blood pressure was 155.

The obstetrical history, together with the blood pressure readings of a number of cases of eclampsia and the pre-eclamptic condition, will be given, showing collapse after rapid delivery plus veratrum viride, collapse after veratrum alone, collapse* after rapid delivery without medication.

*The broader meaning of shock to include collapse is to be taken.

Case 1. Rapid Delivery Plus Veratrum Viride.—M. R., age thirty-two, v-para, admitted to Bellevue Hospital February 13, 1910, with the history of having had six convulsions. She was comatose, her body was edematous, her urine on catheterization was very dark in color and contained a large amount of albumin. She was placed on the table and prepared for delivery. At 2.15 p. m. the blood pressure was 205. Ten minims U. S. P. tincture veratrum were given by hypodermic injection.

On examination the cervix was found to be nearly dilated, a version was performed and a live eight months child easily extracted at 2.23 p. m. The cervix was torn into the lower uterine segment, but there was very little bleeding. Five minutes after the delivery of the child the blood pressure was 150 and 10 minims more of the veratrum viride were given by needle. At 2.30 p. m. the placenta was delivered by Crede. The total blood loss was estimated at 7 ounces. Only a few drops of chloroform were necessary for the entire proceeding. At 2.35 p. m. the pressure was 150. Saline irrigation was given by rectum and two drops of croton oil were placed on



Case 1.—Blood pressure chart. Fall of 105 mm. in 35 minutes. Rapid delivery plus veratrum.

the back of the tongue. At 2.50 or twenty-seven minutes after the birth of the child the blood pressure had dropped to 100, pulse 116.

At 3.30 p. m. the patient was constantly moving her limbs and crying out. The blood pressure was 104, pulse 116. Caffeine stimulation and rectal irrigation were given, but the patient died at 4.30. The child lived and left the hospital in good condition ten days later.

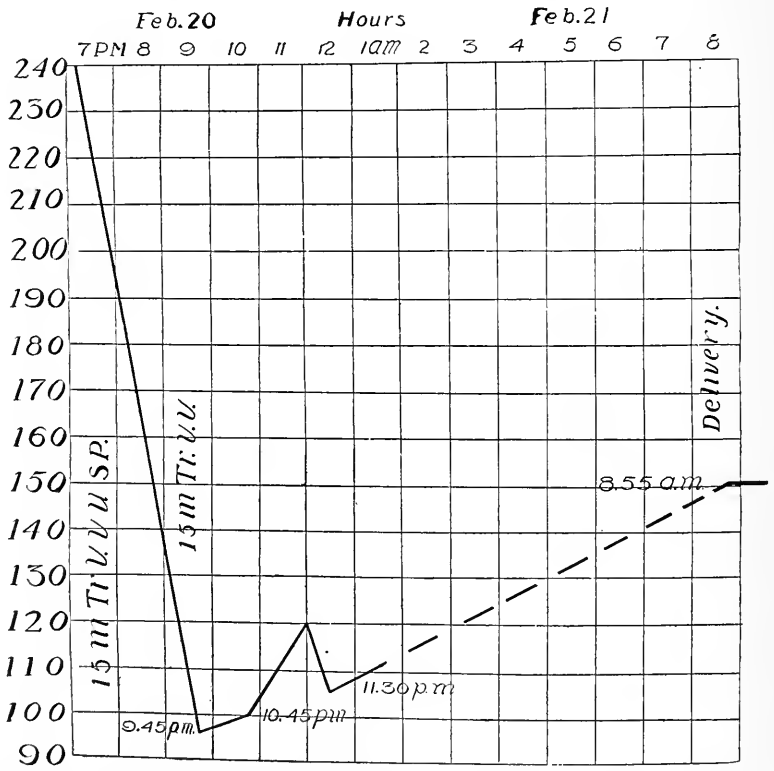
Case II. Collapse From Veratrum Viride Alone.—The patient, M. H., aged twenty-one, i-para, was a pre-eclamptic admitted to Dr. Edgar's service at the Manhattan Maternity February 20, 1910. She suffered from headache, edema, nausea and vomiting, and there were albumin and casts in the urine.

At 7 p. m., February 20, 1910, her blood pressure was 240. Fifteen minims of U. S. P. tincture veratrum were given by hypodermic injection and the dose repeated at 9 p. m. When the second dose was given she complained that her nausea was more intense, but the pulse was not slower. At 9.45 p. m. the patient became unconscious, had constant nausea, frequent vomiting and constant drooling of frothy saliva. Her extremeties were cold, the pulse rate was 60, respirations were 45 and her pupils were dilated. The blood pressure was 95 at 9.50 p. m., a drop of 145 mm. of Hg. in three hours. She was in evident collapse and treatment was energetic and constant. Strychnia gr. 1/20 was given by hypodermic injection, the extremities were raised and tightly bandaged, every ten minutes for a few doses 10 minims of adrenalin 1-1000 were injected into the muscles of the leg, and saline irrigation was

given by rectum. With the idea of depressing the vomiting center, morphine, gr. 1/8, was given at 9.55 and repeated in one-half hour.

At 10.30 p. m the blood pressure was 105, pulse 80, and the patient seemed better and could give rational answers to questions. At 11.15 p. m. the pressure was 105 and the pulse 80 The bandages were removed from the lower extremities and the pressure at once dropped 10 mm. Application of bandages to one of the extremities raised the pressure to 112. About midnight all drooling of saliva ceased and the patient fell into a sleep. Labor began at 5 a. m., February 21, and delivery occurred at 8.50 a. m. It took twenty-five minutes to resuscitate the infant, which was premature and died a few hours later. Directly after delivery the blood pressure was 150.

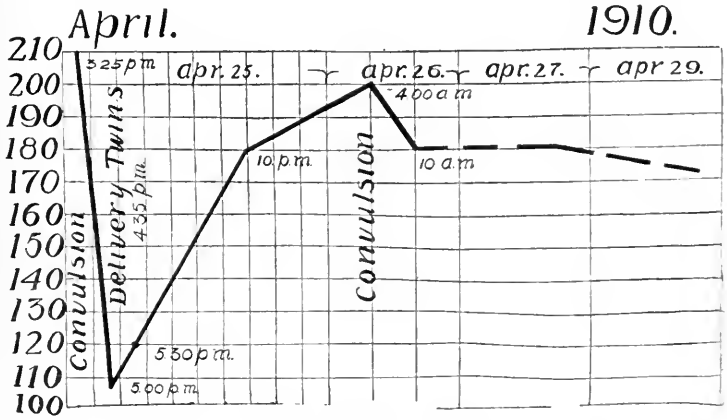
Case III. Collapse with Delivery Alone.—A. R., aged twenty-five, i-para, was admitted to Bellevue Hospital April 25, 1910, with the history of having had one con-



Case II.—Blood pressure chart pre-eclamptic state. Veratrum viride. Fall of 145 mm. Hg. within 3 hours.

vulsion. At 3.25 p. m., shortly after admission, another convulsion occurred and the blood pressure taken at that time was 210. She was placed on the table and prepared for delivery. The cervix was fully dilated and a breech was presenting with the membranes ruptured. At 3.45 p. m. a child was delivered by breech extraction and another head engaged at once. A half hour later a second child was delivered after an easy median forceps operation. The placenta was expressed by the Crede method after twenty minutes. There was only the ordinary amount of blood lost and the cervix was not lacerated. There was a medium tear of the perineum, which was sutured at once. The anesthetic was ether, only a small amount being given. Both children lived.

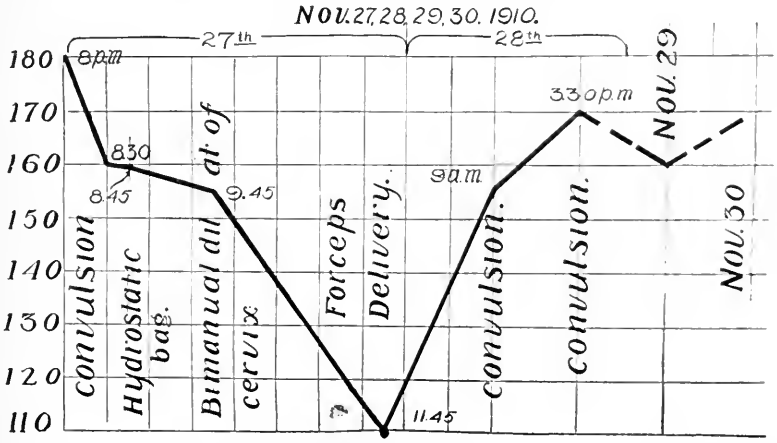
At 4.35 p. m the blood pressure was 108, pulse 128. Hot saline irrigation was given by rectum and the patient returned to bed. At 5.30 blood pressure was 120, and at 10 p. m. it had reached 185, pulse 112. Croton oil was placed on the back of the tongue, nitroglycerin, grains 1/50 gr., was ordered every three hours and a hot



Case III.—Blood pressure chart. Delivery (twins) in eclampsia. Temporary drop of 102 mm. of Hg. within 1½ hours.

pack was given. At 4 a. m. the blood pressure was 200 and the patient had four convulsions in rapid succession. At 10 a. m., April 26, the pressure was 180 and was unaffected by treatment, remaining at that figure for several days. The patient had a good recovery

Case IV. Collapse After Operative Delivery.—C. K., aged twenty-five, i-para, was admitted to Bellevue Hospital November 27, 1910, with the history of having had



Case IV.—Blood pressure chart. Eclampsia operative delivery. Fall of 70 mm. of Hg. in 3½ hours.

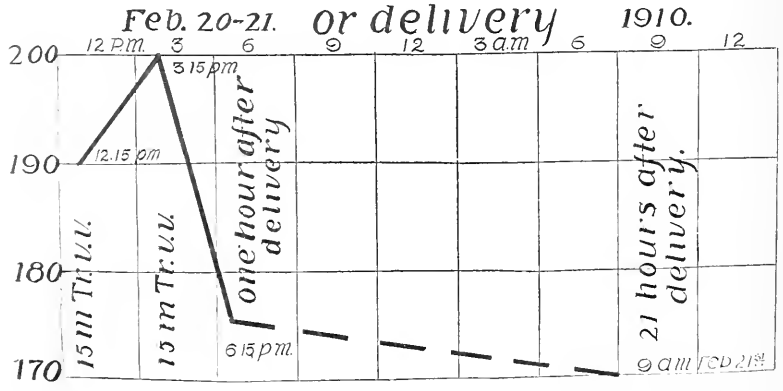
one convulsion. On admission at 8 p. m. she was maniacal and practically blind. She had marked edema and a catheterized specimen of urine showed the presence of considerable albumin and some blood. Her blood pressure was 180.

On examination she was found to be at term with the cervix two fingers dilated and rigid. A three-inch bag was inserted and forceful pains began at once, expelling the bag in one hour. At 9.45 the blood pressure had dropped to 155. Her cervix was bimanually dilated and high forceps were applied. At 11.25, after a difficult opera-

tion, a full-term child was born. While its heart was beating for half an hour it could not be resuscitated. There was a marked second degree laceration of the perineum, which was repaired at once. The placenta was delivered by Crede at 11.45 p. m. and at that time the blood pressure had dropped to 110. The patient was returned to bed and continuous rectal irrigation was begun. Croton oil was placed on the back of the tongue.

The following morning at 9 o'clock the patient was still blind and very nervous. Blood pressure was 155. Nitroglycerine, 1/50 gr., every three hours, and hot packs were ordered. At 9.30 a convulsion occurred and again at 3.30. At the time of the last convulsion she was sweating profusely and her blood pressure had risen to 170. The nitrogen partition of her urine showed a low total output with low ammonia and high rest nitrogen percentage. Her eyesight improved daily and her edema diminished, although her blood pressure remained in the neighborhood of 160. She was discharged on the fifteenth day.

Case V. No Apparent Action, Moderate Dosage of Veratrum Viride.—G. R., aged thirty, ii-para, admitted to Dr. Edgar's service at Manhattan Maternity, February 20, 1910, was eight and one-half months pregnant. She had headache, nausea, vomiting and albumin and casts in her urine. Labor was induced by means of a hydrostatic bag. At 12.15 p. m. her blood pressure was 190 and she received fifteen minims of U. S. P. tincture of varatrum by needle. After two hours the pressure was



Case V.—Blood pressure chart. Pre-eclamptic. Little effect on blood pressure of veratrum.

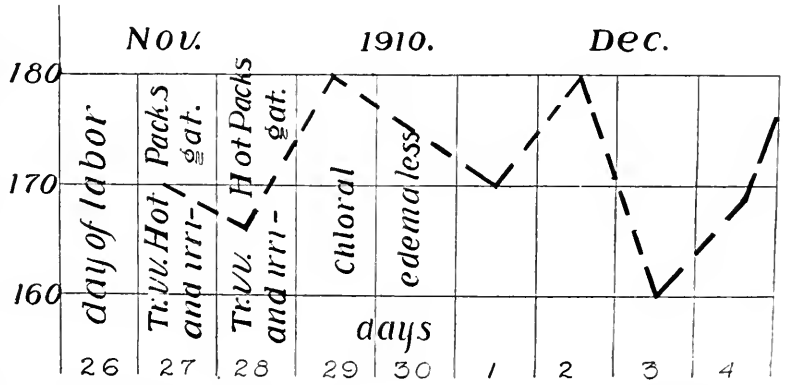
200. She was in active labor and at 4.15 a second dose of the same amount was given. Delivery of a living child occurred at 5 p. m. At 6.15 p. m. the blood pressure was 175. The following day it was 170. She had an uneventful recovery.

Case VI. No Apparent Action, Moderate Dosage of Veratrum Viride.—M. S., aged thirty-one, iii-para, admitted to Bellevue Hospital November 25, 1910. She was somewhat edematous, but went into labor and was delivered November 26. One day after delivery she was markedly edematous over the entire body. Urine examination showed the presence of considerable albumin and epithelial and granular casts. Her pulse was rapid and showed considerable tension, but no other symptoms were observed. The blood pressure was 170 and with the idea that post-partum convulsions were imminent the tincture of veratrum U. S. P. was given in 15-minim doses. Hot packs, hot saline irrigation and magnesium sulphate were given at different intervals during the next two days and she received in all 150 m. of veratrum. The blood pressure was 168 on November 28, and hardly moved from that level for several days. The diaphoresis and diuresis were copious and the edema diminished rapidly.

Case II shows very clearly the pharmacologic action of veratrum. The dose used was 30 minims and the preparation came from a well-known drug firm and was labelled "Tincture Veratrum Viride." It may be that by accident the drug clerk substituted Norwood's for the official tincture, but this is unlikely, for case V received a like amount of the same preparation on the same day with little effect. Even if this were Norwood's tincture the dose would not be so large as that advised by Hirst (15 m. of Fl. ext.) or that of Gillespie of Cincinnati quoted by Zinke (50 to 120 m. of Norwood's tincture).

This patient may have been very susceptible to the drug, but, whether or no, as the argument is based not on the dose but upon the physiological effects, it becomes evident that the drug produces an extreme degree of shock, not only appreciated by a tremendous drop in blood pressure, but also by other constitutional symptoms.

Pharmacologists agree that veratrum has a collapse action, that it is very similar to aconite and that its action is eventually a depression of the vagus, vaso-motor and other medullary centers. Crile (5) has shown conclusively that the low blood pres-



Case VI.—Blood pressure chart. Little effect of moderate dosage of veratrum.

sure of surgical shock is due to central vaso-motor depression. Cases III and IV show well-marked pictures of surgical shock, and these patients had received no medication. It will be readily seen how the prognosis in these two cases might have been changed by superadded collapse produced by veratrum given in full doses. Case I is an apt illustration of results produced by a combination of these procedures. Following the operation and one dose of the veratrum the pressure fell to 150 and remained at that point for at least fifteen mniutes. The second dose having been given, the pressure again dropped 50 mm., placing the patient in absolute collapse.

The blood pressure observations in these cases certainly show a marked hypotension immediately after delivery, and if it is deemed desirable to lower the blood pressure in this disease, the best method would be the delivery of the child, but the question comes up as to the advisability of reducing blood pressure in these cases. The system is nearly overcome by the circulation of some toxin or toxins and it is an open question as to whether the rise in blood pressure is not one of Nature's protective mechanisms. We all know that in chronic kidney disease it is a part of the protective mechanism, for any attempt to reduce the pressure below a certain level is promptly accompanied by an increase in the other symptoms of the disease.

If the blood pressure is to be lowered why not dilate the splanchnic area without depressing the medullary centers? Dilation of this area is accompanied by an anemia of the brain, which would be very desirable. Wallace and Ringer (6) and also Matthew (1) have shown that nitroglycerine in 1/50 gr. dose will depress the blood pres-

sure 11 to 14 per cent. within five minutes when given by mouth and that this action begins to take place within two minutes.

Together with the nitroglycerine 2 gr. of erythrol tetranitrate should be given because its action begins when the nitroglycerine action wanes and the depression of the blood pressure is continued for about three hours. These drugs are especially active in high blood pressure, and a single dose of each of them would bring the high pressure seen in these cases well within the limits of safety.

Many authors state that under veratrum given in full doses no further convulsions occur. It is quite reasonable to suppose that this is true, for under a condition of profound shock convulsions are not apt to occur under any circumstances.

The prominence given the convulsions and the high blood pressure is apt to be at the expense of the legitimate therapeutic indication; that is, the elimination of toxins. Elimination from the skin by the hot pack, from the kidneys by hot rectal irrigation, and from the bowels by means of active purgation, should find a much more prominent place in the treatment.

Cragin and Hulls' (8) work on the detrimental influence of chloroform in eclampsia takes away another of the old standards and it seems very opportune to protest here against the common custom of allowing the uterus to relax and bleeding to occur after delivery. Until the blood pressure index of the shock caused by the delivery is taken at a period an hour after labor, all of the patient's resources should be conserved. It is exceedingly easy to insert a cannula into a vein and allow a controlled amount of blood to flow at any time that this method of treatment may be deemed advisable.

As this is entirely a clinical paper, the author greatly regrets the small number of cases reported. As these cases are not of frequent occurrence and as so much is written at the present time concerning the beneficial action of veratrum viride, it was considered advisable to give the results of the year's work.

CONCLUSIONS.

1. Rapid emptying of the uterus in eclampsia frequently produces a decrease in blood pressure amounting to 100 mm. of Hg., causing a condition of collapse or shock.

2. Veratrum viride given to its full physiological effect may cause a drop of 145 mm. of Hg. in the blood pressure, producing shock.

3. The administration of veratrum viride combined with emptying the uterus in eclampsia may produce such profound shock that the patient cannot recover.

4. While it is probable that the high blood pressure of eclampsia is one of Nature's protective mechanisms, still if it is deemed advisable to lower it, nitroglycerin and erythrol tetranitrate should be used, for their action is on the peripheral vessels and not on the medullary centers.

5. One of the best means of temporarily lowering blood pressure in antepartum or intrapartum eclampsia is the emptying of the uterus.

The blood pressure estimation was made with the Stanton wide cuff instrument. The cases were from the service of Dr. J. Clifton Edgar at Bellevue Hospital, with two exceptions, which were from his service at the Manhattan Maternity.

I am indebted to my friend, Mr. Frederick Wengenroth, for the drawing of the charts.

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PROTEIN METABOLISM IN LATE PREGNANCY AND THE PUERPERIUM.*

J. R. Murlin, Ph.D. and H. C. Bailey, M.D.

This work was undertaken with a view of studying the metabolism in the last month of pregnancy and determining the value of changes shown by the partition of the nitrogen of the urine, as an index to the pre-eclamptic or the eclamptic state.

There has been tendency during the past few years to associate more closely the conditions known as pernicious vomiting of pregnancy occurring in the early months, toxemia of the middle period and eclampsia at or near term (1). A similar condition of liver necrosis has been found to exist in some cases of each of these conditions, and this has given rise to the theory that the symptoms are due to faulty metabolism. It is thought that the liver is unable properly to transform the nitrogenous bodies delivered to it by the portal circulation.

This theory has obtained so firm a foothold that to-day many obstetricians consider it an additional reason why chloroform should not be used as an anesthetic and its use limited in normal labor, for it has been shown that this drug produces a degeneration of the liver-cells and therefore might prove to be synergistic to a condition of toxemia which is already present, but the symptoms of which are latent (2).

If eclampsia is due to changes of protein metabolism, the fact should be demonstrated by examination of the divided nitrogen of the urine and such partitions should offer the most certain forewarnings of impending convulsions. Many believe that the nitrogen partition does offer just such an index, and accept a high percentage of ammonia or of rest nitrogen as evidence of serious disorder. When present with vomiting in the early months an increase of the ammonia nitrogen to 10 per cent. of the total nitrogen has been accepted by some as an indication to empty the uterus (3).

We wish to call attention to the fact that high ammonia and high amino-acid does not prove an alteration in the metabolism even if the urine which furnishes the high amounts is acid at the time of the examination. It is possible, too, that partitions of the nitrogen after a series of convulsions may give entirely different results than before the attack began (4). We shall present facts concerning the normal limits of the divided nitrogens in the last month of pregnancy and show what little reliance may be placed on single determinations as an indication of the pathologic conditions.

METHODS.

In the course of this study we have examined the urines of normal women, of pre-eclamptics and of eclamptics.

The urines were collected for twenty-four-hour periods. They were placed in large

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bottles containing 10 c.c. of 10 per cent. thymol in chloroform and left in a cool place either in the ice-box or out of doors (in the winter months). The collection was made with the greatest care possible and a number of the cases were under the immediate care of one nurse.

As soon as the urine was received at the laboratory the acidity and the gravity were taken and albumin tests were made. It was then diluted to 2,000 c.c. with distilled water. The ammonia was done by the Folin method, running the air stream from seven to sixteen hours. The creatinin was determined by the colorimeter and the creatin was ascertained by a slight modification of the Folin method. The urine was then stored in the ice-box, and usually not later than the second or third day the total nitrogen was determined by the Kjeldahl and the urea of the Benedict methods. The amino-acid or, more properly, the "formol-titrating" bodies, were determined directly in all but two cases by the new method of Henriques and Sörensen (5); in these two (Cases 5 and 8) by a new method devised by Benedict and Murlin (6). The purim nitrogen was determined by a modification of the Walker Hall method (7), which consists of the absolute determination of the nitrogen in the silver purin precipitated. The details of the method will be published shortly.

Physiologic significance of the Different Nitrogen Fractions.

Urea.—The clinical significance of this fraction, taken alone, is greatly overestimated. Not less than two-thirds of the urea present in a twenty-four-hour sample of urine has its origin in the nitrogenous materials ingested as food within the same twenty-four-hour period. It is an old fallacy which dies hard, that the protein materials eaten as food are all first built up into tissue proteins, and that all the nitrogenous waste products result from the daily wear and tear of the tissues. But it has been abundantly proved by the Voit school that the amount of nitrogenous waste products in the urine depends on the amount of nitrogen in the food and that any nitrogenous material absorbed into the blood beyond the needs of the moment is immediately eliminated in the urine. How useless then to determine the urea either by weight or as percentage without taking into account the amount of nitrogen in the food! For illustration, in our third normal case, on the second day of our observations, there were 19.1 gm. urea in the urine, which that day had a volume of 1,470 cc. The next day the amount of urea was 19.6 gm. and the volume 722 c.c. On the former day the urea was 1.3 per cent. of the entire urine and its nitrogen was 79.5 per cent. of the total; the next day it was 2.7 per cent. of the urine and its nitrogen 88.5 per cent. of the total. The metabolism as judged by the absolute amount of urea was almost exactly the same; as judged by the percentage amounts it was very different on the two days. From every clinical sign both of these days were entirely normal, the difference being due to a change from meat to milk in the diet. It is obvious that the absolute figures are a much more reliable index in such cases than percentage.

Urea.—Two conditions (8) are known to reduce the amount of urea below the normal: (1) acidosis, which calls for ammonia to neutralize acid bodies formed in metabolism or otherwise introduced into the body, and (2) retention of nitrogenous materials for growth of the body as a whole, or for growth of some part normally or pathologically. In the dog it has been shown that the growth of the fetus, uterus, mammaries, etc., is sufficient in the last weeks of pregnancy to show an appreciable effect on the absolute and relative amounts of nitrogen appearing as urea in the urine (9), and there is every reason to believe that a careful balance of the total intake and total output of nitrogen together with determinations of the urea would show the same thing in pregnant women. Many satisfactory balance studies have been made (10), but nobody has yet combined the two. What we wish to insist on,

however, is that low urea at such a time is not an untoward sign. In fact, it is just what is to be expected; for the studies on the dog referred to above have shown that it is that portion of the nitrogen which would have been excreted in the urine as urea that is especially retained for formation of the fetal tissues. When the urea is low, both absolutely and relatively, as in late pregnancy, convalescence, etc., the other nitrogen fractions, which tend to remain constant in the absolute sense under all conditions, are necessarily higher in the relative sense (11).

Ammonia.—This fraction is always present in the urine for the purpose of neutralizing the acids which are formed in the oxidative processes of the body. For example, sulphur is oxidized to sulphuric acid and phosphorus to phosphoric acid. If there is sufficient alkali furnished by the ash of the food, very little ammonia is required to render these acids harmless; but in so simple a change in the diet as substitution of potato-starch by rice, Sherman and Gettler (12) have shown recently that the ammonia increases in the urine from 0.1 gm. to 0.15 gm., because rice furnishes a more acid ash. Janney (13), on the other hand, has been able to drive the ammonia out of the urine almost completely by administration of sodium bicarbonate with the food. In diabetes, in which the imperfect combustion of fat leads to the production of acid bodies, the ammonia rises very high, and in starvation the same sort of change, though not to so great a degree, takes place. When the ammonia rises in the absolute sense, it is always at the expense of urea, so that even in severe acidosis the urea-plus-ammonia fraction of the nitrogen may be exactly the same as normal.

High ammonia in the absence of an acidosis means decomposition of the urine after the urine reaches the bladder. We wish to lay emphasis on the possibility of decomposition in the bladder as well in as in the bottle. A catheter may be perfectly sterile and the hands that use it may be sterile also, but if the catheter pushes ahead of itself up the meatus a plug of mucus containing bacteria capable of decomposing urea and if the urine contains albumin, high ammonias will be found, in spite of the most careful preservation of urine in the bottle.

In support of this we may cite the following experiences: two women (primiparæ) in perfectly normal puerperium carried high ammonias throughout the confinement period; both had been catheterized for several days following delivery. Another woman (tertipara) under the same physician, nurse and dietary regulations, who was not catheterized but passed her urine spontaneously after carefully cleaning the parts, went through the puerperium without showing a single high ammonia. Again, a case of eclampsia (Case 8 of this series) showed after several days' use of the catheter, on a certain day, 1.06 gm. nitrogen as ammonia. The next day, after thorough irrigation of the bladder twice in twenty-four hours with a saturated solution of boric acid, the ammonia dropped without any change in the food or other condition of the patient to 0.460 gm. Finally, we may mention catharsis as another cause of high ammonia percentages. For example, with Case 2, catharsis and irrigation of the colon on the twelfth day of our observation caused so great a reduction in the amount of food absorbed and metabolized that the urea fell from 9 gm. to 4.5 gm. The ammonia was 0.57 gm. and 0.58 gm., respectively, but rose in percentage of the total nitrogen from 9.8 per cent. to 17.3 per cent. How many of the so-called high ammonias which have been thought sufficiently grave to indicate emptying of the uterus have been obtained after catharsis?

Creatinin.—Folin was the first to show that this nitrogen-containing body is the most constant in amount for any given individual of any of the nitrogenous constituents of the urine. On a meat-free diet the absolute daily output is found to be proportional to the muscular development of the individual (14).

Creatin.—It is generally conceded to-day that creatin in the urine of an adult on a meat-free diet, means undernutrition, i. e., undernutrition as regards energy-

producing constituents of the diet, especially carbohydrates, or destruction of muscle (15).

Purin Nitrogen.—The purin bodies are end-products of the metabolism of nucleoprotein. It is usual to separate the purin bases, xanthin, hypoxanthin, adenin and guanin from uric acid, but the general significance of all of these bodies is the same, and we have determined them together as total purin nitrogen. The presence of much purin nitrogen in the urine signifies the ingestion of meat or some richly cellular animal food. On a meat-free diet the purins are generally low unless actual destruction of tissue cells is going on.

Amino-acids.—This fraction has figured considerably in the literature of pregnancy-urines in the last ten years, because the presence of amino-acids in large amounts has been thought to indicate a deficiency in the power of the liver to split off the nitrogen from the amino-acids which enter the portal circulation as the final products of digestion. Without entering into a lengthy discussion of this point we may say at once (1) that the amino-acid nitrogen is not synonymous with rest-nitrogen or undetermined nitrogen; it must be determined separately. (2) If the amino-acids were present in the urine of pregnancy in increased amount the fact would not prove deficiency in the de-aminating power of the liver, for they might originate from enzyme action in the placenta, in which case they would not necessarily pass through the liver before reaching the kidney. (3) A higher percentage of amino-acid nitrogen in late pregnancy is to be expected in view of the lower percentage of urea nitrogen caused by the retention on behalf of the fetus.

Undetermined Nitrogen.—Adding together all these fractions—urea, ammonia, creatinin, creatin, total purin and monamino-acid nitrogens—there yet remains a small fraction undetermined. Of course, we must recognize the fact frankly that what remains may be error. If an error of only 0.5 per cent. should occur in each of the several determinations made and all of the errors were by chance in the same direction, there might be an apparent undetermined nitrogen fraction of over 3 per cent. Assuming, however, that in most instances such errors would counterbalance each other, we find left in almost all of the urines we have examined an undetermined fraction of from 3 to 9 per cent. A very small part in some cases is due to albumin; in a few urines we have determined what Henriques and Sörensen call the polypeptid nitrogen, i. e., extra amino-acid nitrogen after digestion of the urine with strong hydrochloric acid. The indications are that this would account for a considerable part of what we called in our tables the "undetermined nitrogen." Henriques and Sörensen have found such a fraction in normal urines.

Normal Women in the Last Months of Pregnancy.

At Bellevue Hospital (from the service of J. Clifton Edgar), the ante-partum women were carefully examined, and three were selected who were in the last month of pregnancy and free from abnormalities. They were placed in a separate ward under the care of a nurse whose main occupation was the supervision of their diets and the collection of their urines. The women did no work and were practically in a condition of rest during the course of the observations.

On the first two days and the fourteenth or last day of the observation period, they were kept on the regular ward diet which contained meat. From the second to the eighth day they received a diet of whole milk, bread and butter and milk sugar. An effort was made to keep the calories supplied proportional to the requirements of the individuals. On the eighth and ninth days, in additiont to the above diet, they received daily fifteen grains of thyroid extract. From the tenth to the fourteenth day they were fed a larger quantity of carbohydrate, and on the thirteenth day they received a gram of pure creatin with their food.

The pulse, respiration, weight and blood-pressure were taken daily. The weights remained practically the same during the entire period except for fluctuations of about a pound. That they were well nourished and that the children's weights did not suffer was shown by the fact that within three weeks they all had children weighing, respectively, 9 pounds, 4 ounces; 9 pounds, 8 ounces, and 7 pounds, 2 ounces. (Table 1.)

Each of the three women remained very constant in the total amount and the proportional amounts of the nitrogen excreted throughout the observation period. A glance at the table of averages (16) of the eleven days shows that one patient (Case 2), excreted about one-half as much nitrogen as either of the others. She weighed less and was fed more exactly in proportion to her physiologic requirements than were Patients 1 and 3. The urea was low, and the ammonia was normal in amount. The percentage figures for the ammonia were high throughout. Six of the eleven days it was over 10 per cent. and one day following catharsis it rose to 17 per cent. The average ammonia figure was 10.5 per cent. The monamino-acid and the undetermined nitrogen were low in amount although the percentage figures were high. The percentages are to be explained by the higher retention of nitrogen in these case than in the others.

The average of the thirty-three normal urines shows a urea figure somewhat lower than in the non-pregnant, while the other fractions are slightly higher. On the whole, there is very little difference.

PREECLAMPTIC WOMEN.

Examinations were made of the urine of three preeclamptic women, one of whom developed convulsions two hours after the birth of her child.

Case 4.—Patient, B. P., a multipara, aged 34, admitted October 9, 1911, complained of headache, dizziness, sleeplessness, nausea, bad vision and swellings of her legs. She had a trace of albumin in her urine and her blood-pressure was 160 mm. She was placed in bed and kept on a milk diet for seven days. She had a number of hot packs and many of her symptoms improved, although the headache persisted for a number of days and the blood-pressure went as high as 195 mm. on October 16. On October 14, 15 and 16 she received in addition to the milk diet 15 grains of thyroid extract. She was delivered on October 26 and had a normal puerperium.

Case 5.—Patient, M. A., a primipara, aged 25, admitted to the Bellevue Hospital School for Midwives, October 7, 1911, had headache, nausea and slight edema of the extremities. There was a trace of albumin in her urine and the blood-pressure was 130 mm. of Hg. She was placed on a milk diet and on October 18, 19 and 20 she received in addition 15 grains of thyroid extract. She was delivered October 24 and had a normal puerperium.

Discussion of Cases 4 and 5.—While both these women presented signs of impending eclampsia, there were no abnormal changes in the partition of the nitrogen. The largest amount of ammonia excreted by B. P. (Case 4) was 0.97 gm. on October 19. The highest percentage excretion was on October 12, 0.43 gm. (5.9 per cent.). The highest ammonia excreted by M. A. (Case 5) was 0.86 gm. (5.5 per cent.) on October 12. The preceding day the ammonia was 0.39 gm. or less than one-half and yet the percentage was about the same (5.1 per cent.). The amino-acid nitrogen was low in total amount and in percentage. Table 2 shows that the average composition of eight urines in the one case and of eleven in the other was perfectly normal.

TABLE 1.—NORMAL WOMEN

Case Number	Amount Grams								Per Cent. Total N						
	Total N	Urea	NH_3N	Cr_1N	Cr_2N	Purin	Formol Titr.	Undet. N	Urea	NH_3N	Cr_1N	Cr_2N	Purin	Formol Titr.	Undet. N
1. Average 11 days.....	11.50	9.0	0.74	0.35	0.11	0.29	0.505	0.53	78.2	6.4	3.0	0.98	2.5	4.3	4.6
2. Average 11 days.....	5.08	3.60	0.536	0.218	0.037	0.239	0.239	0.19	70.86	10.55	4.29	0.7	4.7	5.1	3.8
3. Average 11 days.....	9.57	7.79	0.496	0.346	0.097	0.34	0.335	0.27	81.4	5.1	3.5	0.1	3.4	3.3	3.2
Average 33 urines.....	8.71	6.77	0.59	0.30	0.08	0.28	0.36	0.33	77.7	6.7	3.4	0.9	3.2	4.1	4.0

TABLE 2.—PREECLAMPTICS

Case Number	Amount Grams								Per Cent. Total N						
	Total N	Urea	NH_3N	Cr_1N	Cr_2N	Purin	Amino-Acid*	Undet. N	Urea	NH_3N	Cr_1N	Cr_2N	Purin	Amino-Acid	Undet. N
4. Average 8 days.....	11.67	9.78	0.52	0.42	0.17	0.15	.....	0.63	83.8	4.4	3.6	1.5	1.3	...	5.4
5. Average 11 days.....	**10.75**	8.73	0.47	0.29	0.20	....	0.05	1.01	81.2	4.4	2.7	1.8	...	0.5	9.4

*By new method of Benedict and Murlin.

PREECLAMPSIA AND ECLAMPSIA.

Case 6.—Patient.—A. K., aged 21, a primipara, was admitted October 20, 1911, to the School for Midwives. On October 23, she complained of headache and dizziness and she had some edema about her ankles. There was a trace of albumin in her urine and her blood-pressure was 130 mm. Hg. Her urine was collected for three days. On the fourth day, October 28, the patient went into active labor and delivered herself at 8:30 p. m. During labor a specimen of urine was collected. Two and one-half hours after delivery she had a convulsion and during the next forty-eight hours she had in all eighteen convulsions. Between the convulsive attacks she was stuporous or semi-conscious. On the third day she became maniacal, but returned to normal mental condition on the fifth day. From then on her recovery was uneventful.

The patient was catheterized twelve hours after labor, not having voided in the meantime. A specimen from this urine was sent to the laboratory and the urine passed for the next few days was collected. Specimens of urine were obtained for three days before the convulsions, a few hours before, the first urine passed afterward and a number of subsequent specimens. They present a very interesting and instructive group of nitrogen partitions of the urine of preeclampsia and eclampsia (Table 3).

Discussion of Case 6.—The first three urines show normal urea and ammonia amounts and percentages. Urine 4, taken a few hours before the first convulsion, and Urine 5, taken twelve hours after are remarkably alike and contained the same amount of ammonia. Urine 6 was the first twenty-four-hour specimen after the convulsions began and shows a normal urea and ammonia output. Urine 7, the second twenty-four-hour specimen, after the convulsions contained 1.38 g.m. of ammonia, or 31 per cent. of the total nitrogen. The following days the ammonia excretion was more than doubled (3.20 gm.), although the percentage of the total nitrogen was only 21 per cent. This ammonia cannot be considered as representing an ordinary acidosis, for the urine gave no reaction for acetone or diacetic acid. We have also made an estimate of the amount of ammonia which might be required to neutralize any lactic acid formed. The largest amount found in the urine of an eclamptic patient by Zweifel (4) was 0.621 gm. reckoned as zinc paralactate. This would correspond to about 0.080 gm. ammonia. During the next two days (Urines 9 and 10) the ammonia figure remained high. We have reasons, which will be fully presented in a subsequent paper, for thinking that all these high ammonias, very common in the puerperium, are due to decomposition in the bladder caused by use of the catheter.

In this case therefore the urine gave no indication of an impending attack and the ammonia was low at all periods up to the second twenty-four hours following the first convulsion.

INTRAPARTUM AND POSTPARTUM ECLAMPSIA.

Case 7.—Patient: E. M., a primipara, aged 23, admitted to Bellevue Oct. 19, 1911, at 11 a. m., was in labor and the membranes had ruptured. At 4 p. m. she had a convulsion which was repeated three times before her delivery at 8:10 p. m. The patient was seven and one-half months pregnant and had a tuberculous hip with the right thigh fixed in adduction. Because of the small size of the child the forceps was applied and an easy delivery effected. The child weighed 3 pounds 14 ounces and left the hospital with the mother November 13.

Before the forceps operation the blood-pressure was 185 mm. Hg. One hour post partum it had dropped to 167 mm. A convulsion occurred three hours after delivery and another at eight hours. The following day the patient was comatose, cyanotic and had many moist rales of edema in the chest. Her blood-

TABLE 3.—POST-PARTUM ECLAMPSIA (CASE 6)

Urine No	Date	Total N	Urea N Gm	NH_3N, Gm.	Cr_1N, Gm.	Cr_2N, Gm.	Total Purin N, Gm.	Formol Titr. N, Gm.	Undet. N, Gm.	Urea N, Per Cent.	NH_3N, Per Cent.	Cr_1N, Per Cent.	Cr_2N, Per Cent.	Total Purin N, Per Cent.	Formol Titr. N, Per Cent.	Undet. N, Per Cent.
1	Oct. 23-24	8.06	6.02	0.45	0.31	0 0	0.31	0.41	0,66	74.7	5.6	3.9	0,0	3.9	5.1	6.8
2	Oct. 26-27	11.09	8.37	0.62	0.43	0.0	0(?)	0.78	0.94	75.5	5 6	3.9	0.0	...	6.6	8.4
3	Oct. 27-28	7.90	6.31	0 46	0.31	0.0	0.28	0.40	0.14	79.8	5.2	3.9	0.0	3.5	5.0	2.6
4	Oct 28 (labor)	0.45	0.34	0.03	..	. .	....	...	....	75.6	7.5	...	...	...	...	...‡
5	Oct. 29 (14 hours after delivery).	0.41	0.30	0.03	....	...	....	....	....	73.2	7.0	...	...	..	...	...†
6	Oct. 29-30	4.87	3.61	0.11	0.25	0.0	....	....	....	74.1	2.3	5.2	...	...	...	...*
7	Oct. 31-Nov. 1	4.42	2.29	1.38	0.40	0.0	0.08	0.16	0.11	51.8	31.2	9.1	0.0	1.8	3.5	2.6
8	Nov. 2-3	15.11	9.80	3.20	0.51	0.12	0.03	0.37	1.08	64.8	21.2	3.4	0.8	2.0	2.4	5.4
9	Nov. 4-5	15 73	11.07	2.42	0.46	0.08	0.06	0.45	1.19	70.4	15.4	2.9	0.5	0.4	2.9	7.5
10	Nov 5-6	12.41	9.02	2.03	0.36	0 06	0.06	0.31	1.60	72.7	16.4	2.9	0.5	0.5	2.5	4.5

* First twenty-four hours' urine after convulsions. † Urine fourteen hours after convulsions began. ‡ Urine just before convulsions.

TABLE 4.—ECLAMPSIA

Case No.	Date	Amount Grams			Per Cent	
		Total N	NH_3N	NH_2N†	NH_3N	NH_2N
7	Oct. 20-21, 1911	12.01	2.22	0.38	18.5	3.2
	Oct. 21-22, 1911	6.51	1.10	0.18	16.9	2.8
	Oct. 24-25, 1911	8.26	0.32	....	3.9	...
	Oct. 28-29, 1911	11.96	0.53	0.34	4.4	2.8
	Nov. 4-5, 1911	5.88	0.20	0.15	3.4	2.5

† Method of Henriques and Sörensen.

Case Nd.	Date	Amount Grams			Per Cent.	
		Total N	NH_3N	NH_2N*	NH_3N	NH_2N
8	May 2-3, 1912	8.74	0.60	0.06	6.8	0.7
	May 3-4, 1912	16.27	0.63	0.17	3.8	1.0
	May 4-5, 1912	12.80	0.60	0.27	4.7	2.1
	May 5-6, 1912	12.03	1.06	0.43	8.8	3.6
	May 6-7, 1912	8.52	0.46	0.12	5.4	1.4

* By new method of Benedict and Murlin.

pressure was 110 mm. She received stimulation and saline irrigation by the drop method. At 5 p. m. she regained consciousness and rapidly improved. Ten days post partum her blood-pressure was 125 mm. Hg. and she had only a trace of albumin in the urine. No urine was obtained in this case before delivery.

Case 8.—Patient.—M. C., aged 18, a primipara, admitted May 1, 1912, had been delivered in the morning and had had a convulsion shortly afterward. She arrived at the hospital about eight hours after the first convulsion. During the night or for the first twelve hours after admission, she had between forty and fifty convulsions. Between the attacks and for thirty-six hours after she was deeply comatose. She received croton oil, 2 minims, by mouth and two injections of ¼ grain of morphin. A hot pack was given every six hours and high colonic irrigations every three hours. On admission her blood-pressure was 204 mm. On the second day she was maniacal and remained so for forty-eight hours. She had a rapid convalescence. There was considerable albumin in her urine and also hyaline and granular casts. The urine was obtained by catheter for a number of days.

Discussion of Cases 7 and 8.—E. M. (Case 7) had six convulsions in all. She was profoundly ill and her recovery was hardly looked for. The first twenty-four-hour urine showed high ammonia 2.22 gm., or 18.5 per cent. Only one-half this amount was present on the second day, 1.10 gm. (16.9 per cent.), and on the fifth day it had returned to normal, 0.32 gm. (3.9 per cent.).

M. C. (Case 8) had over forty convulsions. Her urine was collected by catheter and many bacteria were found by microscopic examination. On May 7 the bladder was washed twice during the twenty-four hours and as a result the ammonia dropped from 1 gm. to 0.4 gm.

While Case 7 would tend to show that an acidosis resulted from the convulsions, Case 8 with seven times as many convulsions showed normal amounts of ammonia and amino-acid nitrogen (Table 4).

Summary and Conclusions.

From the study of the partition of the nitrogen of 100 urines, it would seem that the ammonia and the amino-acid plus undetermined nitrogen fractions may be as high or higher in normal women in the last month of pregnancy than in women who have preeclamptic signs or even eclampsia itself. Convulsions themselves do not necessarily produce a condition of acidosis. High ammonia following an eclamptic attack, we believe, is often due to decomposition of the urine within the bladder from contamination by the catheter. The following conclusions may be drawn.

1. The average of many determinations shows that the nitrogen fractions of the urine in the last month of pregnancy are but slightly different from those in the non-pregnant.

2. Normal women in the last month of pregnancy may have an ammonia-nitrogen as high as 17 per cent. (after catharsis) and a combined amino-acid and undetermined nitrogen of 10 per cent.

3. Percentage figures alone are deceiving and of little value, for the total nitrogen is dependent on the amount of food absorbed and this is affected by intake, nitrogen retention, catharsis, etc.

4. With all the clinical signs of preeclampsia, the nitrogen partition may be normal even up to and twenty-four hours following the development of convulsions.

5. The nitrogen partition as an evidence of metabolic processes cannot be said to offer an index to the preeclamptic or the eclamptic condition. Alterations in the structure of the liver, and finally in the metabolic functions of this

organ may be, for all that the urinary analysis shows, the result of the toxemia which ultimately leads to eclampsia, rather than the cause of the toxemia.

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A REPORT ON A CASE OF OSTEOMALACIA, WITH A REVIEW OF THE AMERICAN CASES.*

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Osteomalacia is such a rare disease and its etiology so shrouded in mystery that it is the duty of those who by chance meet such a condition to report the same with as much detail as the patience of the reader will permit, with the hope that in the future some lucid thinker and investigator may, from the mass of facts and theories, distill a drop of truth which will crystallize into a knowledge of the cause and treatment of osteomalacia.

The patient was brought to the hospital in an ambulance and admitted in September, 1911. She was born in Sicily, thirty-nine years of age, and had resided in New York for ten years; height 4 feet, 5½ inches; para x, oldest child eighteen years of age, youngest child four years and living; two other children living, aged eight and six; five children died from various diseases. All previous labors were easy and no instruments used.

Upon admission to the hospital patient was pregnant at term, having been in labor for twenty-four hours. Cervix fully dilated, membranes ruptured, fetal heart about 120 and regular, pains were frequent, but with no advance of the head which presented, child in L. O. A. position.

On examination the pelvis was found flattened by a marked projection forward of the sacrum, the internal conjugate was made out to be about 10 cm. and the true conjugate estimated at 8 cm. The pubic arch was very much narrowed, the rami being almost parallel. The child's head was felt to be very soft, though of normal size with moderate molding. Being unable at this time

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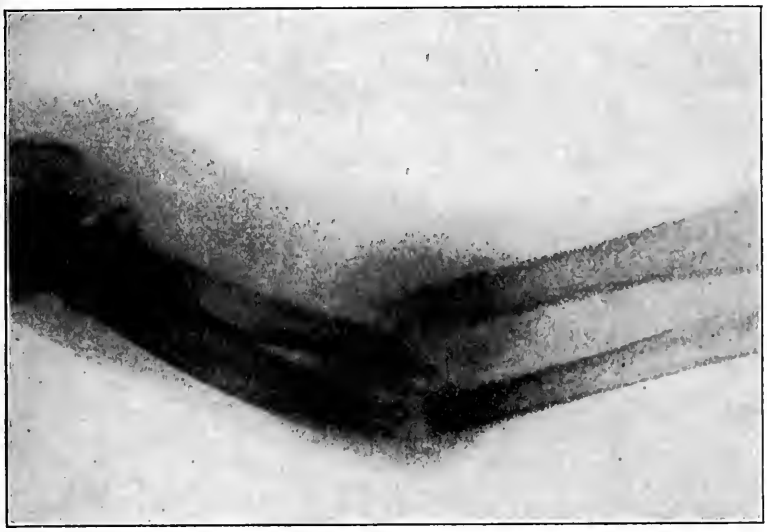
to obtain any further history than already mentioned and not suspecting osteomalacia, we were at a loss to explain how a woman with such a pelvis could have had nine easy labors without instruments, but finally concluded that probably the marked softening of the child's head permitted excessive molding and spontaneous birth. At this time I thought also of other cases of deformed pelves, in which a Cesarean section had been strongly advised, but which when left alone delivered themselves spontaneously.

As the membranes were ruptured, the cervix fully dilated and the head molded, forceps were applied in the high position and traction used for a few moments, but the head did not seem to advance. At this time we considered the advisability of performing Cesarean section but as the membranes had been ruptured some time and as the fetal heart was now irregular, I did not consider it the part of wisdom to subject the mother to the risk of an abdominal operation



Skiagraph of left forearm, from a case of osteomalacia, produced by patient elevating herself on the arm in bed. The thin cortex present in both bones is also shown.

under these conditions, to save the child, which even now was not in good condition. Estimating that only a slight increase in the pelvic diameters was needed for delivery, pubiotomy was considered; but before attempting this and because the child's condition was becoming worse, forceps were again applied, and as some advance was made with each traction, the instruments were continued and after fifteen minutes of intermittent traction the child was born; and though the fetal heart continued to beat for several minutes, the child never breathed. Upon completion of the patient's history, when we made our diagnosis of osteomalacia, I was very glad that we did not perform pubiotomy.

In the middle of the patient's left forearm was a deformity consisting of a bony mass slightly larger than the forearm and an angulation of thirty degrees from normal. At the site of this deformity, there was a moderate amount of free motion—five to ten degrees. Upon the patient's admission, this was thought to have been the result of an old fracture, but upon getting her history later on, we learned that this had come gradually for the last year, because of the fact that the patient had spent most of this time in bed, and the deformity was due to rising on the arms while in bed.

Previous History.—One year ago the patient began to have irregular pains in the hips and along the legs, and at the same time complained of feeling weak. This pain and weakness continuing over a period of six months she finally had to go to bed and remained there for six months before entering the hospital. The patient's living children are all healthy and well nourished, as is also her husband, and they all live together.

On examination the patient appeared thin, but not emaciated; there was moderate anemia and her spirits were good. She complained of severe pain in both hips, especially the left, upon exertion, even upon being lifted and turned in bed, with great sensitiveness to pressure. This pain was so severe that it was impossible (without an anesthetic) to turn the patient on her side to take pelvic measurements. The left trochanter major was displaced inward and upward, and flexion at the hip-joint was possible only for about eighty degrees; the right hip-joint could be flexed to about ninety-five degrees. There was marked lordosis of spine in lumbar region and the crests of the ilia yielded to pressure.

Pelvic Measurements.—Interspinous diameter, 26 cm., intercristal, 29 cm., right oblique, 20 cm., left oblique, 21 cm., external conjugate, 19.5 cm.

Examination of the urine showed the following features: amber, cloudy, specific gravity, 1025, albumin, 0.1 per cent., by Esbach, diacetic acid present, but no casts.

A blood count showed 4,450,000 red cells, 16,000 white cells, 70 per cent., hemoglobin, with a differential count of 82 per cent. polynuclears and 18 per cent. mononuclear cells. No malarial plasmodia found.

An X-ray examination was made at the Gouverneur Hospital.

Osteomalacia is a comparatively rare disease and since the collection of cases reported by Dock in 1895 I have gathered the following from American medical literature.

Case 1.—1895. C. G. Cummiston (Annals Gyn. and Ped., vol. viii) reports a case of osteomalacia in a woman born in Geneva, age thirty-nine, para vii, in whom the condition started eight years previously with pains in both legs, rendering walking difficult. At this time she was treated for chronic rheumatism and the condition became worse with each succeeding pregnancy (v-vii inclusive) though there was some improvement after each labor. Finally the patient was compelled to be in bed all the time, having intense pain in legs, pelvis and kidney region, with violent muscular contractions of legs during sleep only.

Physical Examination.—Abdomen prominent because of lumbar kyphosis pelvis and thorax approximated, slight flexion at knees, some inward rotation of thigh and projecting beak at pubis.

Measurements.—Interspinous, 18¾ cm., intercristal 26 cm., ext. conjugate, 16¾ cm., true conjugate 7 cm. Ascending rami of pubis parallel, tuber ischii separated two finger-breadths, coccyx displaced forward. Movements at hip-joint: flexion at ninety degrees, pain great; abduction very limited, outward rotation ten to fifteen degrees. Shoulder rotation limited, elbows and hands normal. Height 141½ cm. (4 feet 8½ inches).

Treatment.—Castration with considerable improvement. Ovaries normal except few small cysts.

Case II.—1903. Montgomery (J. A. M. A., vol. xli) reports a case of osteomalacia, but the history is so meager and the dementia and paralysis such a prominent factor that we must be doubtful of the correctness of the diagnosis.

Case III.—1904. T. A. Davis (Annals of Surg., vol. xl) reports the first case of osteomalacia in the male in North America; a Norwegian, age thirty-three, in the United States twenty-five years, married thirteen years; three

healthy children. Had been treated for rheumatism of feet and ankles; then fractured humerus from slight fall which fracture refused to unite. A diagnosis of sarcoma made and amputation of the shoulder-joint performed. The blood showed white blood cells 6,800; red cells, 3,704,000. The urine was normal. Pathological examination after amputation showed osteomalacia, and patient was much improved after operation.

Case IV.—1904. Malsbury (International Clinics, vol. 14, S. ii) reports a case of osteomalacia in an unmarried girl about twenty-three years old, never pregnant, whose symptoms began at about sixteen years, with pains in the lower extremities and pelvis and difficult locomotion, sensitiveness to pressure. Presence of rathitic rosary and bow legs marked, myasthenia. The urine was normal. Radiographs showed shortening neck of femur on either side. X-rays pass through bone as if soft tissue. The pelvic measurements were as follows: Interspinous diameter, 22.5 cm., intercristal 23.5 cm., external conjugate 17.5, diagonal conjugate 6 cm.

Treatment.—Panhysterectomy, improved.

Case V.—1905. McPherson (Bull. Lying-in Hospital vol. vii, No. 1) reports the only case of osteomalacia occurring in the service of the New York Lying-in Hospital, up to that date in over 40,000 cases.

Para x, age forty-two, Austrian, and in the United States eighteen years, married twenty-two years. First noticed that spine became crooked, then pains in the back, thorax, groins and legs and unable to walk without assistance. Height 134½ cm. Weight 49 kilos. Pelvic measurements: I. C., 21½ cm., I. S. 22½ cm., R. O. 21½ cm., ext conj. 19½ cm., L. O. 22 cm., diag. conj., 10 cm., true conj. 8 cm. Rami of pubis approximate each other. Urine and blood examinations negative.

Treatment.—Cesarean section by Dr. Markoe; separation of abdominal wound twelve hours later and intestines protruding; resuture; infection of abdominal wound and of uterine cavity; discharged improved on 35th day.

Child died two days after delivery with icterus neonatorum. Autopsy showed atelectasis, general congestion, hyperplasia of spleen.

Case VI.—1906. Oskar Klots (Montreal Med. Jour., vol. xxxv) reports two cases of osteomalacia associated with lipemia, one in the female, one in the male.

a. Female about twenty-four years old, no history of pregnancy or marriage, was operated upon for cystic mass in broad ligament of right side and died after operation. Autopsy: dwarfed girl with bending of left femur and posterior bending of both tibia; bones cut easily and could be pared with a knife, a marked osteomalacic pelvis, the tuber ischii being separated but 2¾ cm., the long bones showed only a shell with honeycombed structure within, containing soft and pulpy marrow. Kidneys showed chronic parenchymatous nephritis. Blood-vessels of lung, liver, kidney, spleen and heart muscle showed fat lipemia.

b. Male, age twenty-four, born in England, in Canada one year. Tibia curved backward; tuberosity right humerus fractured at surgical neck; left humerus broken at surgical neck; all bones soft, easily broken. Autopsy: osteomalacia extending throughout entire osseous system.

Case VII.—1906. McCrudden, Francis H., of Boston reports case in female about eighteen and never pregnant or married. Castration performed, followed by improvement but in one and a half years condition returned and grew worse.

Case VIII.—Stephen Brown (Charlotte Med. Jr., vol. lx) reports a case of osteomalacia in a female, age forty-five, married at fifteen years of age and sterile, menopause at forty-four years.

At thirty years of age rheumatic pains began in hips, followed by pains in bones of left leg, and six months later, broke ankle walking across floor. Two years later broke thigh by some trivial injury; some years later broke left arm. Pelvis flat. This history is very incomplete but it is probably one of a mild case of osteomalacia.

SUMMARY OF CASES REPORTED IN AMERICA SINCE 1895.

FEMALE, SEVEN CASES.

Para	Age	Nationality	Time in U. S.
xi	42	Austrian	Eighteen years
vii	39	Swiss	Not stated
o	23	United States	Life
o	24	Canadian	Life, in Canada
o	18	United States	Life
o	45	United States	Life
x	39	Sicilian	Ten years

MALE, TWO CASES.

Age	Nationality	Time in U. S.
24	English	Canada, one year
33	Norwegian	Twenty-five years

Of these cases reported since 1895, one-half were under twenty-five years of age; one-half were born in the United States or Canada; two were males, and of the seven females four had never been pregnant.

Dock's report included ten cases up to 1895, all of American birth, all females, five single and no children. Four had large families (5 to 10 children), but the disease in question began after menopause. One puerperal case at thirty-five years after birth of fourth child. Dock's own case: Born in the U. S., age twenty-four, ii-para.

Up to the present time, therefore, there have been reported (including our own case) on this continent a total of twenty cases of which only two were male. Of the eighteen female cases, nine or 50 per cent. had never been pregnant; eight or 44 per cent. had four to eleven children, and one had two children.

The number of patients suffering from osteomalacia is probably larger than our figures would indicate because some cases are not reported. J. Whitridge Williams mentions the fact that Hirst has seen three cases in Philadelphia, and that he himself has seen an equal number. Besides the cases not reported, there is probably a considerable number of mild cases that are not recognized, and are treated as chronic rheumatism; and which either improve spontaneously or die of some intercurrent affection or progress to the later stages, when they are generally recognized. In most of the published cases, a previous diagnosis of rheumatism had been made.

Osteomalacia may be defined as a chronic disease of the bones of adults, characterized by pain and associated with progressive muscular weakness, causing a diminution in the amount of inorganic substance of bone and in increase in the organic proportion, leading first to fractures from slight causes and later to increasing softness and bending and deformity of the bones, and ending, as the disease progresses, in complete helplessness and death from exhaustion.

What the underlying cause or causes of this condition may be, it is difficult to determine: poor hygienic conditions, damp dwellings and improper food seem to be accepted as sufficient causes for the disease. But not all the reported cases suffered from insufficient nourishment; and in our own case, all the patient's

family live together and the rest are healthy and strong, and she informed me that she desired to leave the hospital and to return home so that she might have something good to eat!

Multiparity is generally considered the principal factor in the disease and the reports from Europe especially Italy, Austria, Switzerland, and the Rhine Valley would seem to prove it, but of the cases reported on this continent in females, more than one-half had never been pregnant and again the disease is occasionally found in men. Disease of the thyroid and suprarenal glands have been brought forward as a causative factor, but it remains for the future to prove the contention.

The theory of halisteresis, i.e., that an acid thought to be lactic acid, circulating in the blood, dissolves the lime salts of the bone, has had wide acceptance and this belief is due probably to faulty technic in finding lactic acid in the urine of cases of osteomalacia.

The investigations of competent men, however, seem to show that the blood and tissue fluids do not become acid and that lactic acid fed to animals gives negative results. At the present time, the theory of halisteresis has been abandoned.

Another theory of the etiology is that of bacterial infection, but this has few supporters to-day.

Fehling's idea that osteomalacia is a trophoneurosis and dependent upon the int rnal secretion of the ovary, has had wide acceptance and to-day is recognized in most text-books, whose authors recommend castration of the patient to cure the disease. McCrudden of Boston, who has done the most in this country in the study of bone metabolism, especially in reference to osteomalacia, takes issue with Fehling's theory and shows that castration has no effect on the metabolism of normal individuals and reports that his own case, while improved for a short time after castration, relapsed in a year and a half and became worse, and that of Fehling's fourteen cases but six were well three years after operation and two showed temporary improvement and then became worse and the others either died or were lost track of.

The reports of other observers show that a large percentage of patients were not cured by castration and as many of the cases reported as cured by operation were not followed for any length of time, it is very probable that the percentage of cures is less than the figures indicate. Thus far, at least, the histological examinations show no constant changes in the ovaries removed from patient with osteomalacia.

The present idea that osteomalacia is due to disturbances in the metabolism of bone is gaining ground; this theory is based upon the fact that bone like other body tissues undergoes destruction and repair (catabolism and anabolism) and that in normal bone these two processes are equal; whereas, if there is a sudden increased demand for lime salts, be this due to the growing fetus, or to calcification in various tissues, or concretions in the kidneys, the bones undergo increased catabolism and osteomalacia results. This may also ensue from insufficient supply of lime salts in the food to take the place of that used up in normal catabolism.

For details of bone analysis and metabolism experiments in this relation, I would refer to the excellent monograph of McCrudden entitled, "Studies in Bone Metabolism" in the Archives of Internal Medicine, June, 1910.

The pathology of osteomalacia seems to show that the process consists in laying down new calcium-free bone for normal bone that has undergone catabolism.

The symptoms of osteomalacia in the beginning at least are often vague and consist of rheumatic-like pains, generally in the hips, spine and lower extremities; associated with these pains there appears muscular weakness and

frequently ilio-psoas paralysis with abduction of the thigh. The pains are increased by pressure and by movement, so that walking becomes difficult and soon the patient spends most of the time in bed. Soon after the appearance of the disease there is a marked reduction in the patient's height, fractures occur from slight injuries and bending and distortion of the bones ensue (which may be very marked) especially of the spine, pelvis and lower extremities. The prominent beak-like pubis, the projection forward, often extreme, of the promontory of the sacrum, and the parallel descending rami of the pubes, the easily compressible (rubber) pelvis with the attendant pain, make the osteomalacic pelvic picture distinct. The mind remains clear, the knee-jerks are frequently increased, the urinary or blood changes are negative, or at least not constant, there are no visceral changes and the temperature as a rule remains normal.

The symptoms grow worse during pregnancy and may improve considerably for a period after labor, but recur if the patient becomes pregnant again, or the symptoms may grow progressively worse without pregnancy; in general, it may be said that the natural tendency of this disease is to produce death from exhaustion.

The treatment of osteomalacia at the present time is unsatisfactory, though there have been reported cures following simple chloroform narcosis and the administration of chloral and of adrenalin and of thyroid, and with no special treatment. But in most of these cases the improvement lasted only a few weeks or months.

More permanent results have followed hygienic treatment in conjunction with the administration of phosphorus and it is accepted that pregnancy or lactation should not be countenanced. While startling improvement in some cases has followed castration, there have been many failures and we should not place too much reliance upon the operation. Nevertheless, other methods failing, we are justified in performing a laparotomy with this end in view.

The delivery of a living child from a patient with osteomalacia is possible, either spontaneously or with forceps; by inducing premature labor, or by Cesarean section, depending upon the amount of pelvic deformity. However, in this, as in other pelvic deformities, one must not rashly rush into performing that simple, expeditious, though sometimes abused operation of Cesarean section.

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PROCIDENTIA UTERI.

*Suprapubic Plication of Vagina and Conjoined Shortening or Utero-sacral and Broad Ligaments.**

WILLIAM M. POLK, M.D.,

The conclusions formulated by the operation of procidentia uteri suggested at the close of this paper are based upon the inadequacy in my hands of other forms of operation which I have employed from time to time. Convinced of the insufficiency of the lower supports I began with the introduction of the Alexander operation to employ the upper supports. The Alexander operation,

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associated with high amputation of the cervix uteri as a means of diminishing the weight of the organ, was employed for a time; then ventral suspension of the uterus by the fundus; then amputation of the uterus at the cervix and ventral suspension of the stump; then amputation of the uterus with the bringing together over the stump of all the uterine ligaments, broad, round, utero-sacral and utero-vesical, holding them by a kind of purse-string arrangement of the ligatures or by a process of overlapping. The last procedure was reported to this society at its meeting in May, 1909. The others are to be found in the New York Obstetrical Society reports. In addition to these I employed the other methods which have from time to time been suggested by other operators during the last thirty years.

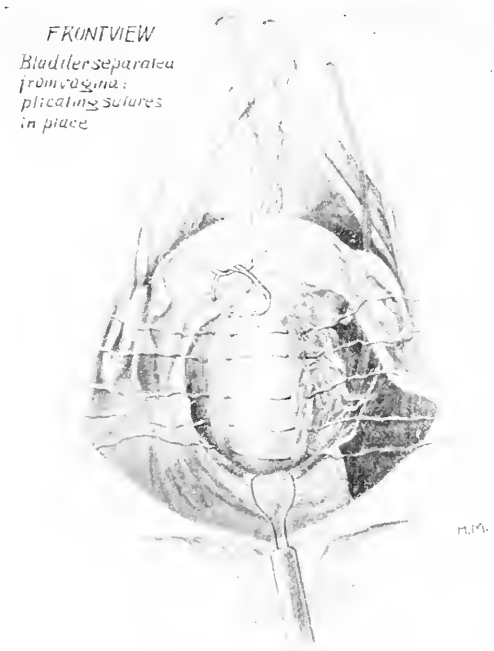
Convinced that the preservation of the circulation and nerve supply, belonging to the uterus, is an essential to the success of any operation for pro-

cidentia, I finally concentrated my attention upon the procedure which I will explain. The essential element is the utilization of all the direct supports of the uterus and vagina; the broad and utero-sacral supports being shortened conjointly, which process may or may not include the round ligament and must be executed so as to spare the blood and nerve supply to the uterus. The utilization of the vaginal supports through plication of its anterior surface (taking in the slack) as far down as the urethra, in fact pulling the sides of the vagina to the center by the plicating sutures; their dealing from below with the inverted vaginal wall, so as to give the utmost possible support to the base of the bladder. The support thus offered to the base of the bladder, together with the utilization of the direct supports of the vagina and uterus, constitute the cardinal features of the operation.

Before submitting to you the details of the operation, I desire to say that the extensive separation of the bladder and ureters from the vagina, which is

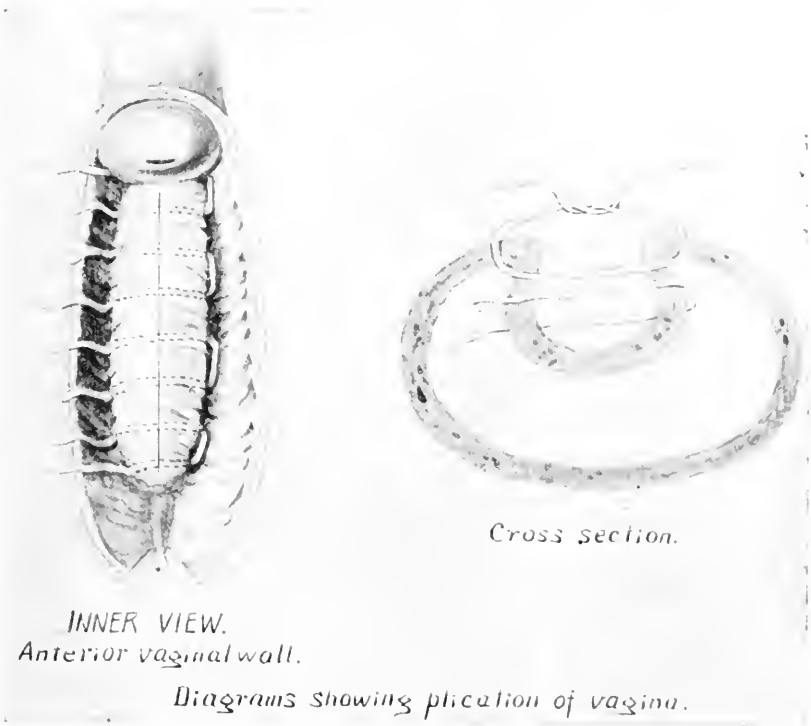
necessary, causes far less hemorrhage and shock that I anticipated; so little, in fact, that in but one case was it a feature of the procedure. I have found also that the more complete the prolapse the easier this separation can be made. I have been unable to complete the procedure in less than one hour and thirty minutes. More facile operators however would find no difficulty in shortening this time materially. Breaches in the perineum and rectoceles may be corrected later, if condition of the patient should forbid it at the time of this procedure.

The operation is as follows: When you have cleansed the vagina paint it over with strong tincture of iodine. Opening the abdomen the pelvis is freed and kept free of all movable coils of intestine. The uterus is now firmly grasped at vaginal junction with a bullet forceps, and the structures put on the stretch forward and upward. Grasp the broad ligament and the utero-sacral fold between the thumb and forefinger; locate the uterine artery where it leaves



the uterus and curves outward towards the main trunk. The thumb being in front and against the uterus, you will have the ureters just at its outer side, behind the artery and extending outward and backward; pass a suture from before backward through the structures held in grasp, entering about half an inch above the artery and about one-third of an inch from the uterus, emerging beneath the utero-sacral fold about one inch from uterus, according to the amount of elongation of this fold. The outline of this suture can be detected by touch, insuring its avoidance of the ureter which is to its outside. Doubling it back above the utero-sacral fold and through the broad ligament below the ovarian ligament it is passed deep into the anterior aspect of the utero-vaginal junction; the loose ends are then temporarily held with a pair of forceps. A suture is passed and secured in similar manner on the opposite side. If these sutures are drawn tight and tied now, the subsequent manipulation upon the vagina may disarrange them. The peritoneal covering is now slit from the

uterus to the bladder, the vagina being held taut upward. Through this opening (it may be enlarged by lateral incisions if necessary) the bladdder is separated from the entire anterior face of the vagina as far as the urethra in extreme cases. This separation is made best with the gloved finger covered with gauze, or with gauze in a sponge holder, the grip of the gauze displacing the tissue with the least risk of injury to important structures. In this separation if one keeps to the vaginal wall, which is recognizable by its smooth and yellowish white structure, the ureters are pushed up and away from this canal, especially opposite the line which the first plicating suture must transfix. This line is as far down as possible upon the anterior surface of the vagina (about opposite the trigone.) Seize the side of the vagina with a bullet forceps, taking



Diagrams showing plication of vagina.

a generous bit, draw it up and pass the suture from without in; repeat this on the opposite side from within out. In this fashion the vagina is plicated from below upward. The number of sutures required depends on the length of the vagina, rarely more than four are necessary; the arteries and veins are tied when necessary. Hemorrhage is rarely a troublesome feature and always easily controlled; rents in the cavity of the bladder are easily corrected, but need not occur.

The next step is shortening the lateral and posterior attachments of the uterus—the base of the broad ligament and the utero-sacral fold. To this end the sutures already in position are drawn taut and tied. This brings all of the attachments included well forward, and shortens them up effectively on each side. The next step is to bring the peritoneum together along the line of your incision, taking in any slack that may exist therein. In this connection the round ligaments and peritoneum may be utilized. That is, the round ligaments

can be caught up about an inch and a half from the uterus, brought together and fastened down at the utero-vaginal junction. This may be done separately or by means of the same suture which encircles the utero-sacral folds and base line of broad ligament. If the fundus then needs to be brought forward you may seize the round ligaments an inch further out and attach that point to the uterus where the ligaments originate. It is now necessary to get the peritoneum which belongs to the anterior face of the broad ligament well down into the utero-vesical space, so as to make the fossa as shallow as possible between the uterus and the bladder. In this way you check the first effects of abdominal pressure; you meet it at the highest point in the field. The abdominal wound is now closed and the vagina becomes the field of operation.

More or less of a ridge will be found upon the central line of the anterior vaginal surface. Seizing this fold at the lowest point with a pair of artery forceps you cut directly into it; the introduction of the director will then enable you to slit it from below upward quite to the uterus without danger of interfering with the sutures that you used in the suprapubic plication. The two folds of the vagina may now be treated as conditions require. You can remove as much or as little as the judgment of the operator may suggest. My own feeling is that little or perhaps none of this fold need be cut away. The surface exposed may be brought together by chromicized gut, which may be put in either separately or continuously as the exigencies of the case require. If preferred this fold may be left intact and its opposite surfaces stitched together by through and through sutures. If the condition of the patient is good and the perineal body needs repair, as it usually does, this may now be done. This closes the operation.

It is quite evident that should we remove the entire anterior wall of the vagina as far down at the urethra, instead of plicating it, the operation would be shortened and simplified. Then one need only bring the cut edges at the sides together at the middle line to bring into play all lateral supports, but the result would be no better than plication gives; in fact inferior, because the column secured by plication gives direct support to the bladder where it is most needed; moreover, if, through any untoward accident or influence, the base of the bladder again descends, it would find adequate covering in the one case, with the other it might not. It is entirely feasible, however, to remove the anterior wall in lieu of plication.

In two of my cases I found the utero-sacral folds so well developed and elongated that I drew them forward along with the base line of the broad ligaments and joined them together in front of the uterus, but it is well to realize that the utero-sacral fold is sometimes a very disappointing structure. Clearly outlined both as regards location, and direction, and well developed, it is readily found, but in some cases it may lack distinctness, and instead of passing directly back toward the sacrum as we hope for, we find it branching off to the right and left and presenting indistinct lines of attachment to the lateral posterior pelvic walls on the two sides. In these cases I have found that the lateral attachments of the uterus as represented in the base lines of the broad ligaments are stronger and more developed than in cases in which the utero-sacral folds have the expected growth. In all of my cases I have met with evidences of shock in but one instance and this was a case that required more manipulation behind the uterus than I have used in my later cases. Such hemorrhages as occur are easily controlled. Most of it is venous, which as a rule has ceased by the time the operation is over. The arterial hemorrhage, if any, is readily controlled. Of course the vital points in the situation are the ureters, but by keeping close to the vagina in separating the bladder, especially as you approach

the lower part of the field, and by carefully inspecting the portion of the vagina at which you pass your sutures, this danger is readily avoided. All sutures except those used for vessels and for closing the peritoneum ought to be of good-sized kangaroo tendon.

A sharp lookout must also be kept as to the ureters when you pass sutures through the broad ligaments and utero-sacral folds, especially in cases in which the latter are ill-defined and incline to lateral rather than sacral attachments. These lateral folds usually run quite close to the ureter as it reaches out towards the pelvic wall.

The conjoined shortenings of the broad and utero-sacral ligaments is easier and more satisfactory than shortening each separately. The fact that the essential fibres of each are continuous at the sides of the uterus justify this step anatomically (1).

I submit seventeen cases as examples, eight within time limit, two years. In every case the uterus rested entirely without the body, or as far out as midway the cervix. One case reported is a prolapse of the vagina and bladder following removal of the uterus years before for procidentia. Treatment by pessary had been tried in all these cases, with failure in each one.

Case 1.—P. E. Age 33. Born U. S. Occupation nurse. Admitted to hospital October 2, 1911. Discharged December 11, 1911. Diagnosis: Displacement of uterus. Complication: Relaxation of pelvic floor. Prolapse second degree. Uterus half out.

Family history negative. Previous history good.

Present illness: Onset several years ago with sudden cramp-like pains in lower part of abdomen. Patient thinks it was caused by a sudden strain on lifting a patient. Next day pain was still present but less severe. Relief followed rest in bed. Since that time patient has been troubled by weight and dragging in pelvis; a feeling of falling out in perineum; occasional nausea and a tendency to tire easily. After a time patient noticed that her cervix was pressing down into vagina and it has at last gotten so low that it protrudes between the labia after standing for some time. Difficulty in voiding urine, sometimes not being able to void without pushing up the uterus. Tendency to constipation. Discomfort gr ater with constipation. No leucorrhoea. Slight loss of weight. Medium height and stout. Organs normal. Sexual organs: External genitals normal. Perineum relaxed. Vaginal walls prolapsed. Cervix conical. Os closed; soft. Uterus low down in vagina; can be drawn down and brought completely outside of vagina through vulva. Adnexa: Negative, except for general relaxation of all ligaments.

October 11, 1911. Operated October 10th. Slept little during night. Considerable pain in bladder region. General condition good. Bladder draining well through retention catheter.

October 12. Doing well. Slight pain. Appetite good. Catheter removed. Has had a few R. B. C. in urine and many W. B. C. Slight pain on voiding.

October 17. Sutures removed. Wound clean and healed. Abdomen strapped.

October 23. Dressing removed from wound. Urine showed some increase in pus. Bladder irrigated with boric acid 4 per cent. q 2 d beginning October 21st. Irrigation followed by argyrol 10 per cent. oz. 2. Up in bed.

October 25. Abdomen restrapped. Bladder irrigations discontinued October 25th. Up in chair 2 hours.

November 6. Ordered up about ward. Condition fine.

November 10. Operation on perineum, November 9th. Very little pain.

November 12. Vaginal douche boric acid 4 per cent. q 2 d. Irrigate perineum boric acid 4 per cent. b. i. d.

November 14. No complaints. No pain. Slight vaginal discharge with slight odor.

December 11. Discharged. Patient examined standing. Uterus in excellent position. No vaginal prolapse.

Operation.—Prepared in usual manner. Incision 5 inches long in mid-line above pubis opening abdomen. Trendelenburg position. No adhesion. Intestines pushed up and packed with gauze roll in keeping intestines free from operating field. Uterus low down in pelvis. Fibroma large as a chestnut on posterior surface of uterus near right cornu. Peritoneum incised transversely between bladder and uterus. Bladder inclusive of trigone separated from anterior surface of uterus and vagina by blunt dissection. Ureters identified and kept free from field of suture by upward and outward traction. Sutures of strong kangaroo tendon passed through lateral wall of vagina on one side, then emerging and passing anterior to anterior vaginal wall to take a similar bite on opposite side, plicating this anterior wall from below upward, from region of trigone to utero-vaginal junction. Five such sutures passed and tied. Round ligaments drawn together in front of uterus and sutured together with Pagenstecher. Peritoneum sutured together over field of operation. Utero-sacral ligaments caught with bullet forceps drawn together and sutured behind uterus with Pagenstecher suture. Small fibroid enucleated and site covered with peritoneum. Abdomen closed as usual. Retention catheter in bladder. Bloody urine oz. 4. Sterile dressing. To bed in good condition. No drain.

April 1, 1912. Reported as doing well.

Case 2.—M. Ph. This patient duplicated in all essentials the preceding case, similar in height, both maidens, and stout, this one of the leisure class, however. Operation the same in all particulars except that here conjoined shortening of utero-sacral and broad ligaments was done and the rectocele and perineum were treated at the time of main operation. March 5th was the date of this operation with a result at this date, April 1st, as good as the best.

Case 3.—D. M. Age 38. Born U. S. Occupation housework. Admitted to hospital October 24, 1911. Discharged December 21, 1911. Diagnosis: Prolapse of uterus. Complication: Double salpingitis. Laceration of pelvic floor.

Family history good. Previous history good. Present illness: During delivery, 17 years ago, patient's perineum was badly torn and symptoms of the present condition began shortly after that time. The first symptoms noticed were a sagging sensation in the perineum and a feeling of weight in the pelvis. She next began to have lumbar backache, pain radiating from the backs of the thighs, and a feeling of something dropping out below. At first this feeling amounted to only a sense of fullness in the vagina, but gradually it grew more pronounced and soon the patient noticed that the vaginal wall seemed to protrude between the labia. This condition progressed until the cervix and finally the whole uterus would protrude from the vaginal orifice whenever the patient would stand for any length of time or strain at stool. During this time the headaches, backaches, and pains in the limbs have been growing steadily worse. Four months ago the patient began to have a profuse brown vaginal discharge after each menstrual period, lasting for about a week. Since onset of the present condition patient has had slight frequency of urination. Sex organs: External genitals, normal. Perineum, extensive old laceration extending to left side of body of perineum. Vagina: Outlet large, gaping. Very roomy. Walls lax and prolapsed. Cystocele and rectocele. Cervix: Fairly large and long in vagina. Uterus: Prolapsed; can be made to descend through vulval orifice. Adnexa: Relaxation of all ligaments.

Operation.—Suprapubic repair of pelvic floor, October 30, 1911. Prepared as usual. Incision 5 inches long in mid-line above pubis. Trendelenburg position. Both Fallopian tubes distended to one and one-half inches in diameter, angulated, somewhat adherent. Tied off at infundibulo-pelvic ligament and at uterine end to tubes and both tubes and ovaries cut away. Vesico-uterine peritoneum divided transversely; bladder separated from anterior uterine and anterior vaginal wall well down. Ureters identified and pushed outward from vagina and upward with the trigone of bladder. Sutures of kangaroo tendon passed through lateral columns of vagina from below upward brought out and tied in front of anterior vaginal wall, thus taking in a reef. Utero-sacral ligaments caught up; threaded with a suture; suture passed through base of broad ligament with Cleveland carrier on either side. The ligament sutured to anterior surface of vagina at level of cervix. Divided peritoneum closed. Round ligaments caught up and shortened by suturing to anterior surface of uterus with Pagenstecher linen. Wound closed in usual manner. Sterile dressings. To ward in good condition. Convalescence uneventful.

November 20. Perineum repaired.

December 21. Discharged. Uterus in good position. Cervix still too long; not involuted.

April 20. Examined standing as before. Utero-vaginal junction in normal position. Cervix still too large but not protruding. Should have been amputated.

Case 4.—M. W. Age 42. English. Occupation, housework. Admitted to hospital May 17, 1910. Discharged June 23, 1910. Diagnosis: Prolapse of uterus. Family history negative. Present history: Ever since birth of first child twenty-six years ago patient has been complaining of sensations of prolapse of pelvic viscera. She has always been of the opinion it was the uterus came down but never came outside. Associated with this she has had backaches of a dragging, bearing down nature but no other pains. Never was treated except once, ten years ago, when she received no relief from pessary tampons and douches. Condition has become progressively worse and for past six months has been feeling weak and run down. Has been troubled with headaches, whitish discharge and occasional vomiting. Comes to hospital on account of prolapse.

General appearance. Rather elderly woman. Poorly developed and nourished. Does not appear acutely ill. Heart normal in size, shape and position. Apex beat in fifth space three and one-half inches from mid-sternum. Sounds of good quality. Not roughened or accentuated. No murmur or thrills. Action good. Arteries rather large but soft. Pulse regular in rate and force; good force and tension. Lungs, chest, fairly developed. Expansion good and equal. No impairment of resonance or changes in breath; sounds, voice or tactile fremitus. No rales heard. Abdomen, soft and relaxed. No tenderness, rigidity or muscular spasm. No masses. Liver, spleen, kidneys not palpable. Muscles, bones, joints negative. Lymph nodes not large. Skin negative. Reflexes normal.

Pelvic examination, external genitals negative.

May 21. Patient's general condition is improved. Constipation has been very stubborn.

May 25. General condition much improved. Intestinal condition better. Locally less congestion. Patient stronger, pulse fuller and better quality.

May 27. Operated. Dr. Polk's operation on pelvic floor. Patient recovered from ether well, with some vomiting.

May 28. Patient has experienced no excessive post operative pain. Had

some cramp-like pains general in region of colon. Passes urine well. Cathartic given.

May 30. No complications. Patient is very comfortable. Bowels move well.

June 3. General steady improvement. Sutures removed. Wound clean.

June 6. General condition shows steady improvement.

June 10. Some temperature. Patient feels well. Wound clean and no tenderness.

June 12. As before. Wound clean but some slight tenderness about upper part of wound and sense of induration deep down. Small opening made in scar but no pocket found. Strip of gauze to keep opening.

June 14. Dressed, gauze taken out and with scissors cut down to induration which is softer. Pus obtained. Finger and scissors enlarge opening and cavity irrigated and pocket packed with gauze.

June 15. Comfortable. Dressed. Wound shows very little discharge.

June 16. Examined by Dr. Polk. Good position. Some induration. No pain. No prolapse.

June 23. Discharged. Condition excellent generally and locally. Good position. No pain; no prolapse. To return for observation.

Operation. Patient and field of operation prepared in the usual manner Incision median about 4 inches long ending 1 inch above symphysis. Abdomen opened and patient placed in Trendelenburg position. Uterus found abnormally movable and all pelvic ligaments stretched. Uterus grasped at cervico-uterine junction by bullet forceps and drawn upward. Peritoneum incised and widely reflected. Bladder with ureters separated from vaginal and uterine walls and retracted. Kangaroo tendon sutures placed transversely across vagina. Whole venous system in region congested and varicosed and bleeding is excessive; controlled by the sutures. Five plicating sutures put in, one or two perforating into vagina.

Peritoneum closed by plain gut sutures running up onto face of uterus and approximating round ligaments at fundus. Sacro-uterine ligaments identified and shortened by kangaroo tendon sutures. Peritoneum and fascial planes closed by catgut and skin by silkworm gut. Last report: Uterus in good position.

Case 5.—B. R. Age 26. Occupation, housework. Admitted to hospital May 26, 1910. Discharged June 24, 1910. Diagnosis: Prolapse of uterus. Complication: Laceration of perineum.

Family history negative. Present illness: Began eight days after child was born, as a pain followed by a prolapse of the uterus. Patient came to Bellevue clinic in December and was treated by pessary which supported uterus until March, when some inflammation set in. Rings were removed and patient put on douches. Since March she has had a yellowish leucorrhoeal discharge. Uterus has been down since that time.

June 6. Operated upon as follows:

Operation.—Patient and field of operation prepared in the usual manner. Incision median about 4 inches long ending 1 inch above symphysis. Abdomen opened and patient in Trendelenburg position. Uterus grasped at cervical junction by bullet forceps and drawn upward. Incision into peritoneum over utero-vaginal junction and peritoneum reflected widely. Uterus and vagina separated from bladder and ureters which are retracted from the operative field. Plicating sutures of kangaroo tendon placed laterally drawing together the vaginal wall anteriorly. Peritoneum closed well up on to face of uterus with catgut, the upper two stitches approximating the round ligaments in front. Uterus held well forward and the utero-sacral ligaments identified. Each shortened

with kangaroo tendon. Perineum closed with catgut as were fascial planes. Skin with silkworm gut. Dressed.

Patient placed in lithotomy position. Incision between rectum and vagina running up to site of old fourchette. Vaginal wall dissected free from rectum and lateral walls of space so left closed with catgut. Skin closed with deep sutures of silkworm gut. Dressed and returned to ward in good condition.

Case 6.—L. L. Age 35. Russian. Occupation, housework. Admitted to hospital November 10, 1909. Discharged January 13, 1910. Married 11 years; 3 children; 1 miscarriage.

Pelvic examination. External genitals: Normal. Bony pelvis: Normal. Pelvic floor: Second degree laceration. Cervix: Median, central, bilateral laceration, bands of scar tissue binding lip of cervix to anterior wall of vagina and to right lateral wall. Uterus: Prolapse in first degree, with cystocele. Adnexa: Normal. Vagina: Roomy. Operation November 15th.

Operation.—Patient and field of operation prepared in the usual manner.

January 13, 1910. Vaginal examination shows little or no discharge. No pain or tenderness except in perineum. Uterus and cervix very high up in vagina. Movable and not tender. Cervix somewhat soft and not patulous. Body of uterus not palpated. Ovaries not palpated; fairly firm bands felt stretching across both fornices laterally, but not making the cervix immobile. Complains of dull, not constant pain over lower abdomen and bladder and in lumbar region of back. No pain on urination. No incontinence. No urgency. Sometimes pain at end of urination. Urination always relieves her pains.

January 8. Complains of dull constant pain in right and left flank. On examination condition is as last noted. Urinary condition improving.

January 10. No change.

January 25. Returned for examination. Cervical stenosis followed second operation and was later broken up. Examined to-day by Dr. Polk. Conditions noted, uterus returned to normal position. Some induration at base of broad ligament on both sides; most marked on left. Patient just complained of pain at menstruation two weeks ago.

April 5. Examined by Dr. Polk as follows: Uterus in good position. Still complains of pains in region of broad ligaments. Tenderness insignificant.

Operation.—Laparotomy and plastic for support of uterus. Patient and field prepared in usual manner. Median incision four inches long made through skin and subcutaneous tissue. Muscle fibres divided. Peritoneum divided. Vagina separated from posterior wall of bladder by blunt dissection, then plicated. Infundibulo-pelvic ligaments shortened. Utero-sacral ligaments shortened; wall of vagina sutured and slack taken up from within abdominal cavity. Peritoneum sutured with catgut; muscle and fascia with plain catgut. Skin sutured with silk. Patient returned to ward in good condition.

Operation on perineum and cervix done December 15, 1909.

April 5, 1910, position good.

April 20, 1912, pregnant six months. Uterus and vagina has remained in good position.

Case 7.—P. D. Age 43. German. Occupation, housework. Admitted to hospital, January 15, 1009. Discharged March 15, 1909. Diagnosis: Prolapse of uterus.

Family history negative. Present history: One year before admission patient says she fell down stairs while carrying a hod of coal and the next day noticed a mass like a ball protruding from the vagina. The mass was not particularly sore or tender but would not remain when replaced in the vagina and has continued to get larger and larger up to present time. Patient has lost

thirty-one pounds in the last four years, since husband's death, but attributes it somewhat to hard and unusual work, as she was forced to support herself. Had a good appetite all the time and states positively that she has had no discharge or pain.

General appearance. Poorly nourished woman of 43, of medium frame, mentality of rather low order. Eyes: Pupils equal, react sluggishly to light and accommodation. Mucous membranes fair condition. Tongue: Slightly coated. Throat and neck: Slightly hyperaemic and considerable enlargement of thyroid gland, especially right lobe is noticeable. Heart: Normal in size and position; no abnormal pulsations seen or murmurs heard. Arteries somewhat thickened. Pulse regular in rhythm and size; small; rate 80. Lungs, negative. Abdomen, wall very relaxed. Striae prominent. No tenderness except on deep pressure on right lower quadrant, where small mass about size of lead pencil was felt. Liver, spleen, kidneys, negative. Skin harsh and dry. Bones, joints, muscles, negative. Lymph nodes, negative. Reflexes, slightly exaggerated. Sexual organs, see pelvic examination. Uterus: About normal in size and when patient lies down is situated low in vagina, but standing or straining, completely prolapses outside vulva.

Operation.—Patient and field of operation prepared in usual manner. Incision made in mid-line between umbilicus and symphysis four inches long and peritoneum opened. Intestines walled off with gauze rolls. Uterus drawn up into abdominal wound. Peritoneum reflection from bladder incised and ureters on each side identified. Sutures of No. 6 plain catgut were then put from the pelvic fascia of each side outside the ureters, then drawing the fascia from each side together under the trigone of the bladder. About four of these sutures were introduced. The uterine arteries on each side and each infundibulo-pelvic ligament were then ligated with No. 6 catgut and uterus cut free from vagina and broad ligaments. Opening in vagina was then closed with No. 4 plain catgut. Then utero-sacral ligament from right side was sutured to stump of broad ligament of left side and utero-sacral ligament of left side was sutured to base of broad ligament of right side, all uniting in one center over stump of vagina and under base of bladder. Peritoneum closed with No. 4 plain catgut, muscle sheath with same and skin with continuous silk suture. Dressing applied and patient returned to ward in fairly good condition.

Case 8.—C. S. Age 39. American. Occupation, housework. Admitted to hospital April 20, 1912. Discharged May 14, 1912. Diagnosis: Displacement of uterus. Complication.

Family history negative. Past history: Operated on at home in November, 1908, for laceration and prolapse of uterus. Personal history: Menstrual: Started at 15; always regular; lasts 4 to 5 days. Moderate daily flow; no pains. Last regular period April 1, 1912, to April 6, 1912. Marital: Married June 28, 1906. Two children, twins, born May 28, 1907. No miscarriages.

Present illness: Patient delivered of twins in May 28, 1907. Instruments used and patient badly torn. She was in bed for about a month. She was up only about three weeks when she noticed that her uterus began to come down and she began to have a discharge. This got gradually worse and she was operated on in November, 1908, and was all right until about July, 1909, when she noticed it coming down again; her discharge began to get troublesome again and it has been very troublesome ever since. Patient's uterus comes out immediately on getting on her feet or soon after and often swells so that she can hardly replace it. She has had irregular bladder symptoms since onset of present trouble. Bowels constipated. Very little backache or headaches. The

vagina, base of bladder and uterus cannot be kept inside the pelvis, unless the patient is recumbent. Uterus and vagina are greatly hypertrophied, and drop wholly out when standing.

Operation April 23, 1912. The vagina was plicated from uterus to urethra. The greatly elongated utero-sacral folds and broad ligaments were shortened by bringing forward the lower half of the latter and the sacral fold of each conjointly uniting them in front of uterus at vaginal junction, and fastening them there. This was accomplished by encircling the folds and the section of broad ligament with a suture, one for each side, tying them together in front, then stitching them down to underlying surface. The plicated fold in vagina was then opened from below, trimmed off, and surfaces coated with catgut. Rectocele and perineum repaired at same time.

Case 9.—F. F. Age 53. Canadian. Occupation, nurse. Admitted to hospital November 14, 1911. Discharged December 18, 1911. Diagnosis: Prolapse of uterus.

General history: Patient is 53 years old; works as a professional nurse. Family history negative. Previous history: Habits good; no alcohol; appetite good; bowels constipated. Usual diseases of childhood. Venereal denied. Always healthy in adult life. Was operated on for alveolar abscess and antrum disease about one year ago. Menstrual history: Menopause at age of 50. Previous menstrual history negative. Obstetrical history negative.

Present illness: Had a rather acute onset. In April, 1911, patient walked the floor for about 20 minutes carrying a heavy, struggling child. The same evening she had lumbar backache and a bloody vaginal discharge. This discharge lasted for about a day, but the backache grew progressively worse. In addition she has had severe occipital headache, pain in the back of her thighs and a sensation of something dropping out below. After standing for a long time or straining at a stool patient says that something protrudes from vulval orifice.

Since onset patient has had frequency of urination, especially during the day, and burning after the act. There is an intermittent, scant, thin, white, vaginal discharge.

Examination shows prolapse of uterus and vagina, the uterus can be drawn outside the vulva but when released about half of it returns.

General appearance: Thin, poorly developed and poorly nourished female of 53. Does not appear acutely ill. Abdomen: Soft; symmetrical; permits of deep palpation everywhere. No masses nor tenderness made out. Liver, spleen, kidneys not palpable. Skin: Somewhat dry; no eruption; no oedema. Bones, joints, muscles, negative. Lymph nodes not enlarged. Reflexes present. Breasts poorly developed; firm; no secretions present. Uterus prolapsed second degree.

Operation.—Patient prepared in usual manner. Incision four inches long in mid-line above pubis. Abdomen opened. Trendelenburg position. Incision through vesico-uterine peritoneum transversely. Bladder separated from anterior uterine and anterior vaginal wall by blunt dissection with periosteal elevator. Anterior vaginal wall plicated by transverse sutures catching one side of vagina, passing anteriorly and then taking in a bite of other lateral wall. Sutures tied. Clamp passed through broad ligaments grasping utero-sacral ligaments, drawing them anteriorly and fastening them to anterior surface of lower portion of uterus in region of cervix with kangaroo tendons. Round ligaments caught, drawn down and sutured to anterior surface of fundus of uterus with kangaroo tendons. Peritoneum not sutured over wound but allowed to drop in place. Abdominal wall closed in usual manner. Anterior vaginal fold slit up

from cervix to urethral orifice and surfaces stitched together formed by sutures introduced in vaginal wall through laparotomy incision. Vagina packed with gauze. Retention catheter in bladder. Sterile dressing. To ward in good condition.

Case 10.—M. D. Age 42. Irish. Occupation, housework. Admitted to hospital September 22, 1910. Discharged November 19, 1910. Diagnosis: Prolapse of uterus. Complication: Old ventral hernia. Lacerated pelvic floor.

General history: Married. Five pregnancies. Venereal denied.

Present illness: Duration about one and one-half years. About one week after leaving Presbyterian Hospital patient felt burning sensation and a feeling as if something had given way in the left lower quadrant. Burning sensation was continuous. Three weeks later patient noticed small lump appearing between labia and this has gradually increased in size. Interferes with walking and makes urination difficult. Has increased frequency of urination. No burning or pain. Has to exert great force to start stream and cannot stop after she has once started. Bowels are always constipated.

Menstruation comes at intervals of six weeks; not painful; flow scant. Has constant sensation of something dragging or falling down about pelvis. No gastric disturbance; slight headache occasionally. No chills, fever or leucorrhoea. Uterus prolapsed. Operation same as preceding case.

Case 11.—E. G. Age 46. American. Occupation, chambermaid. Admitted to hospital October 10, 1910. Discharged December 3, 1910. Diagnosis: Prolapse of uterus. Complications: Lacerated perineum.

General history. Married. Four pregnancies.

Present illness: Duration about seven years. Patient says that off and on for seven years her womb would come down. She had peculiar sensation by which she could feel that it was down, and some times it would protrude outside vulval orifice and would go back into position itself without patient lying or sitting down or any manipulation. Had no pain but interfered with walking. While womb was prolapsed patient would have severe pain on urination. For past three weeks she says her womb has been down and protruding continuously, and patient could not sit down, and urination and defecation were extremely difficult and painful. Uterus prolapsed. Operation same as preceding case.

Case 12.—M. M. Age 50. German. Occupation, laundress. Admitted November 18, 1909. Discharged February 10, 1910. Diagnosis: Prolapse. Complication: Cystocele rectocele.

Family history negative.

Present history: For the last year patient has noticed that whenever she walked or went to stool her anterior vaginal wall would bulge down into the vulva. This is accompanied by severe pain along the sides of her abdomen and in her back. She has frequency of urination but no pain. She is troubled with almost constant frontal and occipital headache. Her bowels are very constipated and she becomes nauseated at the slightest cause; she does not vomit however. Patient feels very weak. Uterus prolapsed. Operation same as preceding case. Result, April 14, 1912, excellent.

Case 13.—E. H. Age 48. Irish. Occupation, housework. Admitted to hospital April 22, 1910. Discharged July 25, 1910. Diagnosis: Prolapse of uterus. Complication: Rectocele, cystocele.

Family history negative.

Present history: Patient was operated on in this hospital and ward eight months ago, and about six weeks after leaving hospital, while at work, she says

her womb came down and was put back with some difficulty. Whenever it is prolapsed she suffers from great pains in back, bearing down sensations, considerable pain and difficulty in urination. She has to wear a perineal support but gets little relief and as condition intereferes with her work she came to hospital for cure. General health has been fairly good. Uterus prolapsed. Operation same as preceding case.

Case 14.—M. D. Age 38. German. Occupation, housework. Admitted to hospital April 25, 1910. Discharged May 6, 1910. Diagnosis: Prolapse of uterus.

Family history negative.

Present history: About three months after patient's last labor eighteen months ago, which terminated in an embryotomy, she began complaining of general ill health and dragging sensations in pelvis as if her insides were falling out. Consulted a doctor, who inserted a pessary and put her on douches. She expelled the pessary shortly after, but continued to treat herself with douches. Had no symptomatic disturbances and felt pretty well except for sensations of prolapse. About one month ago had cough and began to complain of severe dragging pains in back which have persisted. No vesical or rectal disturbances. No discharge.

Pelvic examination: External genitals: Very relaxed, otherwise negative. On coughing or straining, vaginal walls roll out markedly and cervix presents at vaginal orifice. Perineum: Very relaxed, support poor; shows old laceration. Vagina: Ostium gapes, relaxed and roomy with marked degree of rectocele and cystocele; fornices negative. Cervix: Soft, movable, very low down and somewhat anterior. Os patulous. No tenderness. Uterus: Enlarged slightly; well back in pelvis; retroverted and prolapsed about second degree; consistency soft; symmetrical; no tenderness. Adnexa: Negative.

Operation.—Polk's suprapubic operation on pelvic floor. Perineorrhaphy. Patient and field of operation prepared in the usual manner. Incision about four inches long median and ending one inch above symphysis. Abdomen opened and patient put in Trendelenburg position. Pelvis found to contain considerable adhesions about uterus and tubes. Uterus somewhat enlarged and softened and tubes the seat of recent inflammatory changes. Adhesions released. Incision through peritoneum over utero-vaginal junction and peritoneum retracted. Vaginal wall plicated by four kangaroo tendon sutures. Peritoneum closed by catgut sutures extending well up and including the approximation of the round ligaments in front of uterus. Closed ends of the tubes incised and opened. Peritoneum and fascial planes closed by catgut and skin by silkworm gut. Patient put in lithotomy position. Skin between anus and vagina incised and vagina dissected free. Incision goes to site of old fourchette. Lateral walls approximated by catgut suture. Skin closed by deep catgut sutures in antero posterior line. Patient dressed and returned to ward in good condition.

Last report, December, 1911, good result maintained.

Case 15.—F. M. Age 40. American. Occupation, midwife. Admitted, April 26, 1912. Diagnosis: Laceration of pelvic floor. Prolapse of uterus. Family history negative. Venereal denied.

Present illness: Patient comes to the hospital on account of the prolapse of her uterus. She says she was told that she was torn with her first child twenty-seven years ago, but nothing was done and she had no trouble afterwards or after her second child was born, which was twenty-one years ago. Five years after her second child (1896) she again became pregnant and then began to have such severe pains in her lower abdomen and felt so weak that she consulted a doctor, who told her that she had womb trouble from an old laceration

and that she would abort. Patient carried the child for four months by taking very good care of herself and then aborted. Since that time if patient was on her feet much she would have severe pains in her sides and she would go to a doctor and have a tampon put in and rest up and would then be all right for six months or a year. At that time patient could feel the uterus at the opening of the vagina.

Patient has always been constipated and has been troubled with increased frequency of micturition for about ten years.

Patient has been taking a course in midwifery for past six months and had to be on her feet a great deal and all her symptoms have become exaggerated so she came in for an operation. No pulmonary or cardiac symptoms. Has to get up once or twice a night to pass her water as a rule and has severe headaches at times. Sees spots before her eyes and feels dizzy at times. Chief complaints: 1. Prolapse of uterus. 2. Constipation and increased frequency of micturition. 3. Headaches and dizziness. Uterus prolapsed. Can be drawn two-thirds outside pelvis.

Operation.—Repair upper pelvic floor. General anesthesia. Dorsal position. Soap and water preparation. Bullet forceps attached to cervix per vagina. After opening the abdomen the vaginal and supervaginal supports put on stretch by traction on this forceps. Utero-sacral ligaments clearly outlined thereby were then shortened thus: Each seized, successively one inch from uterus, drawn through opening in broad ligament and attached to anterior lateral aspect of utero-vaginal junction. Opening in broad ligament made at inner lower angle of this structure; attachment made several loops of suture. Seizing anterior vaginal wall at utero-vaginal junction this structure put on stretch upward. Bladder separated from anterior face of vagina to trigone. Anterior vaginal wall slit open from utero-vaginal junction to trigone. Lateral walls of vagina approximated along mid-line covering in the incisions. Round ligaments drawn inward and attached to sides of uterus at utero-vaginal junction. Abdomen then closed. Gauze dressing. From below the apposed surfaces of anterior vaginal wall below line of sutures put in from above were then apposed from above downward from uterus to tongue. Repairs of perineum left for future operation. Patient left hospital without repair of perineum.

Case 16.—A. F. British West. Age 40. Occupation, housework. Admitted February 16, 1912. Discharged March 10, 1912. Diagnosis: Complete procidentia. Complication: Prolapse of uterus.

Family history negative.

Present illness: Patient's trouble dates back to the birth of her last child in June, 1907. She was delivered with instruments and she thinks she was torn. No operation was done. Patient was in bed only about ten days and then got up and did her work as usual. Patient was apparently all right for about two years, when she began to have a discharge and not long after she noticed that her "womb was falling." This has slowly and progressively gotten worse until now her uterus comes all the way out. The discharge has gotten worse until at present it is profuse in amount and very foul smelling and is yellow in color. Patient has never had much pain until the last year, when she noticed that if she was on her feet much she would have heavy dragging pain in her back and sides and also headaches. When she gets tired this way it becomes very painful to pass her water. When she pushes her uterus back there is no more pain and she can pass her water easily.

Bowels always constipated. Patient has noticed a little blood in her vaginal discharge for past three or four months and it is increasing in amount. Patient

does not think she has lost any weight. Appetite always good. No cardiac, renal or pulmonary symptoms. Chief complaints: 1. "Falling of her womb." 2. Headaches and backaches if on feet for any time. 3. Vaginal discharge. Sexual organs: Uterus can be drawn outside pelvis.

Operation.—Repair upper pelvic floor. General anesthesia. Dorsal position. Soap and water preparation. Bullet forceps attached to cervix per vagina. After opening the abdomen the vaginal and infra-vaginal supports put on stretch by traction on this forceps. Utero-sacral ligaments clearly outlined thereby were then shortened thus: Each seized successively, one inch from uterus, drawn through opening in broad ligament and attached to anterior lateral aspect of utero-vaginal junction. Opening in broad ligament made at inner lower angle of this structure. Attachment made several loops of suture.

Seizing anterior vaginal wall at utero-vaginal junction this structure put on a stretch upward. Bladder separated from anterior face of vagina to trigone. Anterior vaginal wall slit open from utero-vaginal junction to trigone. Lateral walls of vagina approximated along mid-line covering in incision. Round ligaments drawn inward and attached to sides of uterus at utero-vaginal junction. Abdomen then closed. Gauze dressing. From below: The apposed surfaces of anterior vaginal wall below line of sutures put in from above were then apposed from above downward from uterus to trigone. Repair of perineum left for future operation.

Case 17.—M. O'B. Age 48. Irish. Occupation, waitress. Admitted March 25, 1912. Discharged April 17, 1912. Diagnosis: Displacement of uterus. Retroversion.

Family history: Mother, father and one brother dead. Causes unknown.

Present illness: Patient went to the dispensary because she had "falling of womb." Patient first noticed her womb coming out about three years ago. It did not come all the way out then, but it has gradually come further out until it comes out two or three inches now. It goes back in when she lies down. Patient denies any discharge or any bladder symptoms. She has had bearing down pains in her back since she first noticed that her womb was falling. These are worse now than ever before and are worse when she is walking or working on her feet, being relieved by lying down. Bowels constipated. Chief complaints: 1. Falling of womb. 2. Dragging pain in back.

General appearance: Sparsely nourished; medium stature; dorsal posture; no pain. External movements; eyes normal; pupils equal and react normally. Mucous membranes good color; no patches; tongue clean; throat clear. Heart: No abnormal neck or pericardial pulsation or thrills. Apex, fifth interspace, in nipple line. Area c. dullness, not enlarged. Sounds at apex and base clear. Pulse: Regular in force and rhythm; volume full; tension moderate; v. w. slightly thickened. Lungs: Chest fairly well formed, symmetrical, expansion poor. Fremitus normal, percussion and auscultation negative. Abdomen: Rather sunken but natural contour. No local tenderness or rigidity. No masses. Liver, spleen, kidneys not enlarged, not palpable. Skin: Scars on face and right knee of old accident. Bones, joints, etc., normal. Lymph nodes not enlarged. Reflexes normal. Sexual organs not examined.

April 1. Patient operated on by Barrows. She took ether well and made a good recovery. April 2. Patient in fair condition. She is getting saline per rectum q. 4 h. has had some vomiting. Pulse fair. April 3. Patient is suffering some pain in abdomen. General condition fairly good. Vaginal packing removed. Patient has no complaints. Pulse good. Tongue clean and moist. Quite comfortable. April 4. Condition good. No complaints.

Operation.—General anesthesia. Dorsal position. Soap and water preparation. Abdomen opened by usual median incision. A ventral fixation had been done at a previous operation. The uterus had elongated to about eight inches and was about the diameter of a thumb of median size. The broad ligament was thinned out and the tubes and ovaries were in a position almost parallel to body of uterus. The uterus was cut free and the bladder separated from the anterior surface of the uterus. The utero-sacral ligaments were brought to the utero-vaginal juncture and attached there and the broad ligaments shortened with kangaroo tendon sutures. Abdomen closed. Patient put in dorsal lithotomy position and cystocele repaired. Gauze dressings.

In ending this paper I cannot forbear calling your attention to the advantages to be derived in cases of retroversion of the uterus which cannot be cured in any other manner except by operation. It is well known that a large number of these cases are without symptoms and therefore need no operation. Others can be cured by simply reducing the size of the uterus through curettage and packing; in other words bringing about involution and supporting the organ temporarily with a pessary. There are other cases however, which even though treated by the Alexander operation, or any of its modifications, or by ventral fixation, relapse carrying with them their symptoms of discomfort and even incapacitate nervous and susceptible women. These relapsing cases I believe fail largely through defects in the peritoneal surface of the pelvic floor. Coffey of Portland has suggested an operation which to my mind comes nearer meeting this defect than any other I know of. The transference of the traction about the base line of the uterus to the front, as suggested in this operation for procidentia, has in it, I believe, elements which meet the basic principles required to correct this defect. Separation of the bladder has no place here but narrowing plication of the vagina opposite the cervix, with, or without conjoined shortening of the utero-sacral broad and round ligaments, has something in it worthy of further inquiry.

REFERENCES.

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